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ORGANIC MENTAL SYNDROME WITH PHENOMENA OF EXTINCTION AND ALLESTHESIA

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AND

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NEW YORK

CLINICAL neurologists are rarely inclined to study their patients from a psychologic point of view with the same intensity as they study them from the neurophysiologic standpoint. The clinician readily accepts the observations made by the physiologist on synaptic or neuronal function. He seems to understand these findings because his neurologic orientation has been in this direction. Current teaching stresses that nerve impulses are transmitted in sensory and motor nerves over definite, restricted paths, and in the central nervous system from one point to another through intercellular connections. There is little emphasis on theories which postulate collective action within the nervous system. Such theories have been proposed by Jackson,[1] Goldstein,[2] Lashley,[3] Klüver [4] and others.[5] After reviewing his experimental work, Lashley stated that "behavior seems to be determined by masses of excitation, by the form or relations or proportions of excitation within general fields of activity without regard to particular nerve cells." [3] He further stated that "it is the pattern and not the element that counts." [3]

From Bellevue Psychiatric Hospital; the Departments of Neurology and Psychiatry of the New York University College of Medicine, and the Neurological Service of Mount Sinai Hospital.

1. Taylor, J.; Holmes, G., and Walshe, F.: Selected Writings of J. Hughlings Jackson, edited by J. Taylor, G. Holmes and F. Walshe, London, Hodder & Stoughton, Ltd., 1931.

2. Goldstein, K.: The Organism, New York, American Book Company, 1939.

3. Lashley, K. S.: The Problem of Cerebral Organization in Vision, Biol. Symposia **7**:301, 1942.

4. Klüver, H.: Behavior Mechanisms in Monkeys, Chicago, University of Chicago Press, 1933.

5. (*a*) Walshe, F. M. R.: On the Contribution of Clinical Study to the Physiology of the Cerebral Motor Cortex, Edinburgh, E. & S. Livingstone, 1946. (*b*) Koffka, K.: Principles of Gestalt Psychology, New York, Harcourt, Brace and Company, Inc., 1935. (*c*) Köhler, W.: Gestalt Psychology, New York, Liveright Publishing Corporation, 1929.

However, many clinicians still question the correctness of observations on sensory disturbances which are difficult to explain on an anatomic or reflex basis. Reports of such phenomena as extinction, fluctuation and completion [6]; ring scotoma [7]; polyopia and monocular diplopia [8]; reduction in sensory adaptation time,[9] and spatial disorientation [10] are instances of perceptual disorders which the clinicia is less willing to accept than a report of simple "impairment" or "loss" of sensation.

In previous communications, attention was called to the observation that some patients with injuries in the parietal region were unable to perceive two stimuli at the same time.[11] On simultaneous application of stimuli to the skin on corresponding points on the two sides of the body, these patients felt only one contact, and this on the normal side, and not on the side opposite a lesion in the brain. Routine tests with single stimulations, however, showed that these patients felt all stimuli on either side.[12] This lack of appreciation of the stimulus in such circumstances was called by one of us (M. B. B.) the phenomenon of extinction.[13] Extinction was observed in these patients in all forms, ranging from slight dimming or dulling to complete absence of a sensation. Extinction was found in visual and cutaneous sensation. The

6. Bender, M. B., and Teuber, H. L.: Phenomena of Fluctuation, Extinction and Completion in Visual Perception, Arch. Neurol. & Psychiat. **55**:627 (June) 1946.

7. Bender, M. B., and Teuber, H. L.: Ring Scotoma and Tubular Fields: Their Significance in Cases of Head Injury, Arch. Neurol. & Psychiat. **56**:300 (Sept.) 1946.

8. Bender, M. B.: Polyopia and Monocular Diplopia of Cerebral Origin, Arch. Neurol. & Psychiat. **54**:323 (Nov.-Dec.) 1945.

9. Bender, M. B.: Changes in Sensory Adaptation Time and After-Sensation with Lesions of the Parietal Lobe, Arch. Neurol & Psychiat. **55**:299 (April) 1946.

10. Bender, M. B., and Teuber, H. L.: Spatial Organization of Visual Perception Following Injury to the Brain, Arch. Neurol. & Psychiat. **58**:721 (Dec.) 1947; **59**:38 (Jan.) 1948.

11. Bender, M. B.: Extinction and Precipitation of Cutaneous Sensations, Arch. Neurol. & Psychiat. **54**:1 (July) 1945.

12. Actually, there was no perfect equality of sensibility on the two sides. Disturbances in sensory function of the skin on the affected side could be demonstrated by special methods. Thus sensory adaptation time was found to be reduced in many instances.[9]

13. Bender, M. B., and Furlow, L. T.: Phenomenon of Visual Extinction in Homonymous Fields of Vision and the Psychologic Principles Involved, Arch. Neurol. & Psychiat. **53**:29 (Jan.) 1945. Extinction as used in this, and in previous, communications is defined as the lack of appreciation of one of two simultaneously applied stimulations, although each of them is perceived when tested one at a time. It should not be confused with the term extinction as used by Pavlov (Conditioned Reflexes, translated and edited by G. V. Anrep, London, Oxford University Press, 1927) in his experiments on conditioned reflexes and by Dusser de Barenne and McCulloch [25] in their investigations on electrophysiology of the cortex.

phenomenon was not observed for auditory perceptions in the series of patients with battle injuries of the brain, but Reider has recently reported a case in which hearing became extinct with double simultaneous auditory stimulation.[14]

In the continuation of these studies, we found a patient with suspected Alzheimer's disease who showed extinction in the cutaneous, visual and auditory spheres. In addition to her inability to appreciate two sensations at once, she could not perform seemingly simple tasks when she was confronted with two different aspects of a situation. Goldstein called attention to this disorder and reported several instances illustrating this inability to "perform more than one thing at a time" or "to register more than one concept at a time."[15] In our case of suspected Alzheimer's disease, the disorder in intellectual function seemed to be related to the disturbance in perception, namely, the failure to report the perception of two sensations on double stimulation. Noteworthy was the systematic nature of alterations in function in a patient who showed so-called mental deterioration. A detailed description of the case follows.

REPORT OF A CASE

History.—A white woman aged 39, a stamping machine operator and housewife, was brought to the psychiatric hospital on April 22, 1946, because of her inability to care for herself. She had apparently been in good health until 1943. At that time she had a miscarriage, after seven and one-half months of pregnancy. A macerated fetus was delivered. After this, she was depressed and absented herself from work for about one month. She returned to her job on Jan. 10, 1944. Soon after this, her foreman, who had never previously found reason to complain about her work, which she had performed for the last twenty years, noticed that she was listless and preoccupied. These symptoms varied from one day to another. During this period she was criticized for her inefficiency. She usually responded evasively, complaining that she was fatigued and therefore unable to finish her tasks. At home she informed her sister-in-law that the boss was always pushing her in her job. In January 1945 she was in an automobile accident. She struck her head lightly against the windshield. She was neither unconscious nor hospitalized and had no amnesia for the period.

The decline in the quality of her performance on the job continued into the first half of 1945. In June of that year she complained of dizziness, headache, blurred vision and a fear of working with her stamping machine. At this time her husband, who was in the United States Army at an overseas post, noted that her letters, which had been regular and lucid, became increasingly incoherent. She also became irregular in her correspondence, not infrequently omitting the weekly letters. In August 1945 she stopped writing altogether. Also, in August, her foreman found it necessary to release her from her job because she almost caused a serious accident in the shop. Her condition became progressively worse.

14. Reider, N.: Phenomena of Sensory Suppression, Arch. Neurol. & Psychiat. **55**:583 (June) 1946.

15. Goldstein, K.: The Mental Changes Due to Frontal Lobe Damage, J. Psychol. **17**:187, 1944.

She became preoccupied. Her eating habits, which had always been careful and dainty, became coarse and gluttonous.

When her husband returned from overseas in November 1945, he noticed a change in her personality and appearance. She had become fat and was careless of her personal appearance. She had to be helped to get dressed. She could no longer cook or do other household tasks. She was forgetful and indecisive. She sat for long periods without showing much spontaneous activity. There was no interest in reading or in current news. Occasionally she wet herself.

In February 1946 she was admitted to the Mount Sinai Hospital, New York, for study. There she was found to be disoriented, deteriorated and mildly euphoric. Occasionally she would burst into tears. Her insight and judgment seemed defective. Her gait was hesitant. The plotted visual fields showed concentric constriction in both eyes. The patient frequently became lost in the wards and could not find her own bed. One observer stated the opinion that there was a suggestion of an additional defect in the right homonymous field of vision. An electroencephalogram was reported to be normal. Pneumoencephalographic study revealed slight internal hydrocephalus with a greater amount of air over the left cortical region. The patient was discharged with the diagnosis of presenile sclerosis, or Alzheimer's disease. The patient continued in this state and ultimately was admitted to the Bellevue Psychiatric Hospital.

The past medical history was essentially without significance. The patient was rarely ill. Her childhood development was normal. She completed the eighth grade at the age of 14 years and soon thereafter began to work. There was no previous history of neurotic trends, psychotic reactions or personality changes.

Examination.—The general physical examination revealed a normal condition. The blood pressure was 124 systolic and 76 diastolic. Neurologic examination showed a broad-based, cautious gait with frequent turning of the head and chin to the right side. The associated arm movements were depressed on the right. Coordination and speech were somewhat tremulous but were otherwise normal. The reflexes were active and equal on the two sides. The Babinski sign was not elicited. She exhibited a bilateral positive grasp reflex and slight sucking reflex. Motor power was good. The routine sensory examination showed no consistent disturbances when each part of the body was tested individually and with single stimuli, except for a few changes, which will be described later. With double simultaneous stimulation on the two sides of the body, she reported only one sensation, and this sensation was localized to the left side (phenomenon of extinction). The functions of the cranial nerves were normal except for vision and hearing. These alterations were demonstrated under special conditions, which will be described later. There were no signs of aphasia.

The blood and urine were normal. Lumbar puncture revealed that the spinal fluid was under normal pressure, with a cell count of 1 per cubic millimeter and a normal chemical content. The Wassermann reaction of the fluid was negative. A second pneumoencephalogram again disclosed mild internal hydrocephalus with a greater amount of air over the left cortical area.

MENTAL STATUS

The patient showed profound changes in intellectual functions. She was partly oriented as to place, person and time. Although she realized she was in Bellevue Hospital, she had great difficulty in finding her bed in the ward. Even after her bed was shown her several times, she still could not locate it. She recognized a few physicians and some other members of the hospital personnel with whom she frequently came in contact. She did not know the day or the month.

She sat for long periods beside her bed, never showing any spontaneity or interest in her environment. She answered only when addressed, and usually in single words or simple phrases. Though seemingly congenial, she rarely participated in any activity in the ward or in the conversation which commonly took place among the patients. There was little spontaneous laughter. Her mood fluctuated. In general she appeared bovine, but at irregular intervals she would break into tears. When asked why she cried, she replied: "I guess I am nervous—I can't help it." On other occasions she offered no reason for this behavior.

She ate voraciously, showed bad manners and did not use utensils in her eating unless reminded or prodded. She was careless and slovenly in appearance. There was a uriniferous, and at times a fecal, odor about her body and clothes, which were often dirty, despite frequent changes of linen and hospital gown. Frequently she did not realize that she had wet the bed.

There were many other changes in her mental functions. These will be described under separate headings in succeeding paragraphs.

Despite the apparent mental deterioration, the patient was sufficiently cooperative to give reliable reports. Analyses of her responses to sensory stimuli and of her general behavior showed changes which were consistent from one examination to another. Sensory defects, for example, when found showed a constant pattern and were confined to the right side of the body. Many different functions were implicated. Because of this, it will be necessary to present the data in several sections: (1) disorders in the seemingly simple sensory functions; (2) alterations in motor performance; (3) disturbances in the execution of more complex perceptual tasks, and (4) difficulties in intellectual performances. This subdivision of the material is a matter of convenience. We do not wish to imply that these divisions represent distinct levels, nor do we believe that the disturbances are necessarily unrelated.

Disorders in Sensory Functions.—Disturbance in Cutaneous and Proprioceptive Sensations: Examination of these sensory functions with single stimuli showed mild defects on the right side of the body. She had slight impairment of two point discrimination and point localization on this side. Occasionally, however, she indicated that she was stimulated on her left side, although actual stimulation was on her right side. This form of mislocalization is known as allesthesia.[16]

With the method of double stimulation the defects on the right side became much more apparent. Instead of reporting correctly two stimuli, one on each side of the body, she reported one of two things: (*a*) She claimed that she felt

16. We use the term allesthesia when a stimulus applied on one side of the body is erroneously referred by the patient to the other side. Obersteiner (Obersteiner, H.: On Allochiria, a Peculiar Sensory Disorder, Brain **4**:153, 1882) was the first to describe this phenomenon in 1882, under the term allochiria. However, he did not differentiate the various forms in which allochiria appears. He applied the term allochiria to a condition in which "though the sensibility is retained more or less completely, the patient is not clear, or is frequently, if not constantly, in error as to which side of the body has been touched." On the other hand, Ernest Jones (The Precise Diagnostic Value of Allochiria, Brain **30**:490, 1908) distinguished allochiria, as described by Obersteiner, from a similar phenomenon which he called "allesthesia." In allesthesia the patient has similar difficulty, but the error is "due to incomplete perception of the stimulus and is accompanied by many other failures in perception, such as defective localization." Our patient also showed a relative defect in perception and impaired ability to localize stimuli on the right side.

only one stimulus, and this was invariably referred to the left side (extinction); or (*b*) she said she felt both stimuli on her left side (allesthesia). Extinction was much commoner than allesthesia.

Extinction for pain sensibility was demonstrated by simultaneous application of weighted needles (5 Gm.) in each hand. In these circumstances, the patient reported that she felt the prick of a pin in the left hand but not in the right.[17] The same was true when temperature or touch was thus tested.

The hand was the region in which extinction could be best elicited. When the stimuli were applied to other parts of the limb, such as the forearms, extinction was less evident. It was still less frequent when the tests were performed on the shoulders. Extinction was never manifest with simultaneous stimulation of the two sides of the face or of the two sides of the trunk (chest). In summary, extinction was shown to be present chiefly with stimulation of the hands or feet and less when the more proximal parts of the extremities were examined. Since the extinction phenomenon was found in the acral portions of the extremities, it seemed to have the pattern of sensory disturbances found in cases of cerebral lesions.

The phenomenon of extinction was elicited not only on simultaneous stimulation of the two sides of the body, but on double stimulation of two points on the one (right) side of the body. That is, when the right hand was stimulated at the same time as the right shoulder, the sensation in the hand became extinct; the patient felt the contact in the shoulder, but not in the hand. Extinction was never found with double stimulation of the left side of the body.

Extinction was also found in tests of proprioceptive sensation. Perception of passive movement of the toes and fingers was undisturbed in all extremities when each limb was tested individually. When simultaneous tests were applied, such as passive movements of the index fingers, the patient reported that she felt movement only on the left side. Tests for the sense of vibration similarly produced extinction on the right side.

Extinction was also found in tests of other sensory functions, such as stereognosis, barognosis, graphesthesia, point localization and two point discrimination. The tactile recognition of objects (stereognosis) was defective on the right whenever the patient manipulated another object simultaneously with her left hand. On handling two identical keys, one in the right and one in the left hand, she claimed that she felt a key only in her left hand and nothing in the right, despite the fact that she manipulated the one in the right hand. As soon as the key in the left hand was taken away from her, she felt the key in her right hand. When a pencil was placed in her left hand and a matchbox in her right hand, she manipulated both objects but reported merely that she had a pencil in her left hand. Asked what was in her right hand, she replied that "nothing was there," even though she manipulated the box with her right hand at that very moment. However, she recognized the presence of the matchbox in her right hand as soon as the pencil was removed from her left hand. The same was true for determination of weights. When she was given a 450 Gm. oblong weight in each hand, she claimed she felt

17. Although the patient invariably denied that she felt pain in the right hand on double stimulation, she occasionally withdrew this hand when it was pricked with a pin. This showed that "reflex" withdrawal from pain was preserved even though the patient was apparently not aware of being "stuck." Such dissociation in response to a sensory stimulus, i. e., denial of pain despite withdrawal of extremity away from the stimulus, has been previously described. The most recent observation is that cited by Sandifer (Brain **69**:122, 1946).

a heavy metal object in her left hand and nothing in her right hand. She insisted there was nothing in the right hand, even though the hand was weighted down. On removal of the object from her left hand, she recognized the presence of a weight in her right hand.

When the examiner scratched lightly and continuously the palms of her hands, the patient reported that she felt a "scratching" in her left hand and "nothing" in her right hand. As soon as the scratching was stopped on the left side, she felt the scratching on her right side.

Graphesthesia (perception of figures, such as the number *4* or *3* drawn with a pencil on her skin) was normal when tested in any one part of the body. Graphesthesia became extinct (there was no perception) in the right hand when a simultaneous stimulus (painful, tactile or mobile) was applied to the left side of her body.

Point localization was impaired in the right hand. Two point discrimination was also slightly and inconstantly impaired in the right hand. When these sensa-

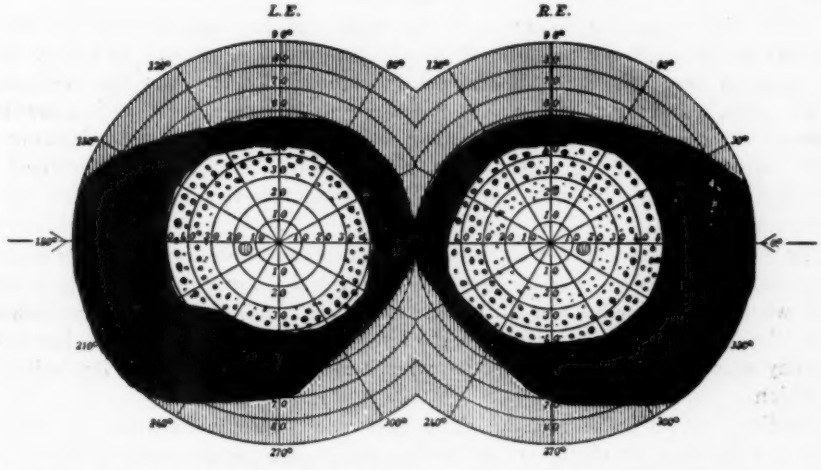

Fig. 1.—Perimetric fields of vision for perception of movement. The solid black indicates the area in which perception of gross movement was absent; the stippled area indicates the region in which the patient inconstantly saw the moving target, thus yielding a variable picture of tubular vision.

tions were examined with the method of double stimulation, she either did not feel any points on the right side or made large errors in the localization and discrimination of the points.

As noted in tests for stereognosis and graphesthesia, dissimilar stimuli produced the same effects as similar stimuli. Application of a tactile stimulus at one point of her body and a painful stimulus at another resulted in extinction of the pain sense. The extinction with dissimilar stimuli was also on the right side and was most apparent in the acral portions of the extremities.

In summary, any of the cutaneous sensory functions tested with the method of double (simultaneous) stimulation showed extinction on the right side of her body. It made no difference whether the stimuli were equal or unequal in strength or whether similar or dissimilar modalities were tested.

Visual Disturbances: The patient had many visual disturbances. The perimetric field of vision was found to be generally constricted (fig. 1). This con-

striction of the visual field was reflected in her general performance. She seemed to miss or ignore objects in the periphery of her field. From her behavior, one gained the impression that she had tubular vision, even more constricted than the perimetric charts indicated. Frequently, she did not seem to see any objects exposed beyond 10 degrees from the macula. However, this was not a consistent observation, for at times she seemed to select objects in the left lower quadrant of the field.

When instructed to fix on a target with one or both eyes while an object, such as a moving hand, was brought in from the periphery toward the center of her field of vision (confrontation test), she recognized the movement of the hand at about 60 degrees on the horizontal meridian in her right field of vision. The result was similar when her left field of vision was tested in the same manner. When the patient was instructed to report what she saw, she stated that she saw a moving hand, and when asked where, she occasionally pointed to her left side, despite the fact that the hand was moving in her right field of vision. This phenomenon is an example of optic allesthesia.[18] This optic allesthesia was never observed when her left half-field was thus tested.

With simultaneous stimulation of the right and the left half-field of vision, the patient reported she saw the object on her left side. Asked to report what she saw on her right side, she replied, "Nothing." There was thus extinction in the right half-field of vision. At times, the patient reported that she saw two objects and that they were both on her left side. This is another instance of optic allesthesia, since the object in the right half-field of vision was localized by her as if it had been in the left field. Visual extinction was found with uniocular or binocular tests.

In summary, the perimetric and confrontation tests showed (*a*) constriction of the fields of vision (tubular fields); (*b*) visual extinction in the right half-field whenever a stimulus was introduced in the left field and (*c*) optic allesthesia, with displacement from the right to the left field of vision. Evidently, her right homonymous half-fields were defective when tested in relation to her left half-field of vision.

Auditory Disturbances: Thus far it has been demonstrated that the patient showed extinction for cutaneous, kinesthetic and visual sensation on the right side of her body when the two sides were compared by means of simultaneous stimulations. Examination of hearing showed similar results. Tests for hearing were beset with more difficulties than other sensory examinations. Audiometric examination by the otologist was attempted, but no measurable data were obtained. Our own crude tests showed that the patient could hear the ticking of a watch with each ear. However, sound localization was impaired. When the source of sound was in the midsagittal plane, she usually localized the sound to the left. She had great difficulty in localizing sound which emanated in her right auditory field. She seldom found the correct source. At times she indicated by pointing with her left hand that the sound came from her left side. This observation is an example of auditory allesthesia.[19] There was no disturbance in sound localization when the left auditory field was tested.

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18. Optic, or visual, allesthesia was described by Fere (L'allochirie optique, Bull. méd., 1888, no. 7) and also by Magnin in 1888 (Allochirie visuelle chez une hystérique hypnotisée, Thesis, Paris, 1889; Compt. rend. Soc. de biol. **57**:58, 1888).

19. Auditory allesthesia or allochiria was first described by Gelle (Gaz. d. hôp. **61**:92, 1888), and later in greater detail by Bonnier (Rev. neurol. **12**:324, 1904).

Extinction of auditory perception on the right side was observed when sound stimuli emanated simultaneously from the right and the left auditory field. Thus, when keys were jingled on her right side and hands clapped on her left side, the patient heard only the clap of hands. There was never auditory extinction in the left ear. When auditory extinction was present, it was found only in the right ear. Again, regardless of whether she reported only one or both binaurally presented noises, she usually indicated that they were on her left side.

Disorders in Movement.—In general, the patient sat or walked with her chin turned slightly to the right, but with her head inclined to the left. The gait was broad based. The arm swing was diminished bilaterally. When told to go to a spot, which required her to turn, she invariably turned to the left side. It was difficult to induce her to turn to her right. She consistently chose to turn to her left, even when obstacles were placed in her path to the left and none to the right. This turning to the left appeared to be more than a "preference" for this side. It seemed as though the patient was pulled to her left side.

There was no motor weakness. The hand grips were of equal strength. In performing the sorting and grouping test or the wool-sorting test, she used chiefly her left hand. On reaching for an object or in pointing, she did so with her left hand. This tendency to use the left hand existed even though the patient was told to use both hands; she used both hands, but within a short period she again used

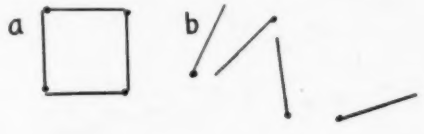

Fig. 2.—The patient's attempt to construct a square from match sticks: (*a*) the square which was constructed for her; (*b*) her attempt to make one.

only her left hand. The patient drew and wrote with her right hand. During her attempts at drawing, it was noted that she often moved her right hand toward the left lower quadrant as though a "force" was pulling her hand in that direction. The examiner frequently moved her writing hand toward the right side of the paper, but she almost invariably displaced the hand toward the left lower quadrant. It seemed that she resisted the movement of her hand to her right side. This pull to the left, or resistance to the right, was apparent in many of her actions. It was noticeable in the various special tests she took.

Disturbances in Execution of Complex Acts.—Apraxia: The patient showed various forms of apraxia. The apraxia, however, appeared to be secondary to disturbances in perception, to right-left orientation and to a disorientation in her visual space. When given a robe to wear, she became confused as to the sides and often put it on backward. She also confused the right and left shoes. Occasionally, in her attempts to write with a pencil, she did so with the rubber end. Although the patient recognized a square, triangle and cross, she could never construct these figures from match sticks.[20] She was unsuccessful even after she was shown how to do it (fig. 2). Furthermore, she could not complete a triangle after two of the sides were constructed for her.

Block Design: The apparent apraxia was also evident when she was tested with the Kohs blocks. She could not place four blocks together to make the simplest

20. The inabiltiy to construct may be termed a constructional apraxia. However, one cannot strictly differentiate visual impairment or agnosia from an apraxia (Mayer-Gross, W.: Proc. Roy. Soc. Med. **29**:1396, 1936).

design. In fact, she could not imitate the arrangement even of two blocks with solid colors. Noticeable was her tendency to set the blocks to her left.

Sorting and Grouping: In the Goldstein-Scheerer grouping tests she failed almost completely.[21] The patient was told and shown how to put all metal objects or all rubber objects together, but was unsuccessful in her attempts. Frequently she did not persist in her task. After a few gestures she stopped her performance and sat placidly. Asked why she did not continue, she said, "I can't see." Further

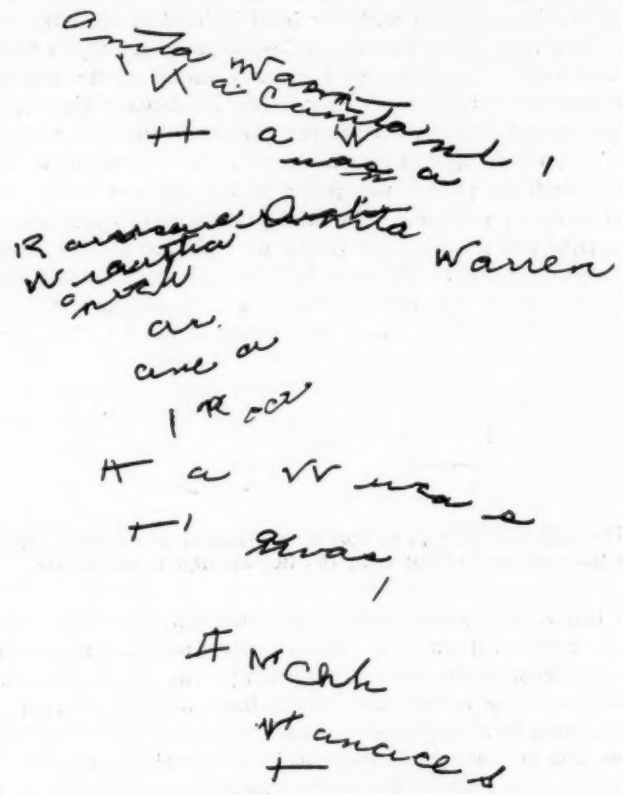

Fig. 3.—Samples of handwriting.

tests showed that the patient had many visual disturbances. She was unable to select an object in a given field even after it was pointed out to her. It seemed as though she lost track of the object designated.

Reading: The patient was able to read single words when these were presented on a white background. She recognized and correctly read "university" when this was the only word on a white sheet of paper. Her reading ability was poor when more than one word or other figures appeared on the paper. Under these conditions she tended to omit and skip from one word to another. Not infrequently, she dropped syllables or picked out only the central letters and disregarded the rest. She selected parts of the whole words, usually the central

21. Goldstein, K., and Scheerer, M.: Abstract and Concrete Behavior: An Experimental Study with Special Tests, Psychol. Monogr. 239, Evanston, American Psychological Association, 1945, vol. 53, no. 2.

syllables. In the word "vibration," she read "brat" or "rat." She had no idea of the total pattern of the word. This particular type of visual disorder was also observed in other visual tests. When shown a picture of a man reading a newspaper and asked to report what she saw, she replied: "newspaper" or "man's hand" or "man." In other words, she did not seem to grasp the total situation which the picture conveyed. She seemed to select one small part of the picture, and not all the parts together as a whole. Conversely, she also had difficulty when instructed to select a part of the whole, such as finding a specific letter in a word. When told to find the letter "e" or "s" in "university," she failed to do so.[22]

Fig. 4.—Samples of spatial disorientation in the patient's writing and drawing. She was directed to copy (*a*) a square and a circle; to write (*b*) #37, (*c*) #68 and (*d*) #68 and (*e*) and (*f*) to make a cross. In her performance, she frequently paid no attention to sequence or proper relationship to the page. Note that the "#37" was written in a vertical direction.

22. Her failure in this, and in other performances may be due partly to her disorientation in visual space. Another factor is that the presence of multiple figures (more than one letter in this case) brought the same visual disability to the fore which she had when she was confronted with multiple words. Although she could read the whole word ("university")—a unitary percept—she was unable to break it down into letters, this act requiring discrimination of one image (letter "e" or "s" or "y") from the rest of the letters. As soon as more than one percept was involved, she failed in her performance.

In her attempt to read a passage which contained several lines, she picked out, at random, one or two words in one or the next line and stopped reading. Her excuse was that the print blurred in her right eye. As a rule, she omitted the upper lines and chose the words nearest the fixation point.

The patient could recognize numbers, geometric figures, drawings and photographs. It was noted that she could not grasp situations suggested by the pictures. Invariably, she selected one or two isolated parts of the photograph, usually in the center or in the left lower quadrant of her field. Curiously, the patient

Fig. 5.—Patient's attempt to copy Wertheimer's Gestalt figures.

recognized words in inverted position. Although she could understand simple and single written or printed words, she could not obey written or printed commands when there were several words in the sentence.

Writing: The patient had great difficulty in writing on dictation or in copying. She never centered her lines, and they were never parallel. Frequently, she wrote at a slant and over other words (fig. 3). In addition, she had a great impulse to write in the direction of the left lower quadrant of the field of vision. The examiner often moved her hand toward the right side of the paper, but slowly her hand shifted back toward the left lower quadrant.

Drawing: The patient had great difficulty in drawing. A circle was drawn but imperfectly, while a square or a triangle was scribbled as a few lines. She could not copy geometric figures (figs. 4, 5 and 6). In drawing a face, she placed the different parts, such as the eyes, nose and ears, in a pattern which was totally unrecognizable (fig. 7). When asked to complete the figure of a face, she could not detect the missing parts, which happened to be the eyes. When told what was missing, she tried to complete the picture by placing the eyes outside the head.

Recognition and Matching of Colors: The patient could name correctly all the primary colors and many of the intermediate shades when a single sample color was presented to her. As soon as there were other colors in the field, she made errors in naming any of them and was completely unable to match them. When presented with variously colored woolen skeins, she successfully named one or another color when it was placed in her central field. On the other hand, when she was asked to select red or green or blue skeins from the entire field, she failed. She also was unsuccessful when instructed to place the various shades of one color together.

Fig. 6.—Patient's drawing of (*a*) a square; (*b*) letters *H* and *X* and a circle, and (*c*) a triangle.

Fig. 7.—Patient's attempt to draw a face. The two dots represent the eyes. The two figures on the right are the ears.

Tachistoscopic Examination: At low speeds (1 sec.) the patient was able to recognize contours of single figures. When there were several figures on the screen, she selected either one in the center or one in the left lower quadrant. At high speeds she recognized the general shape of an object, but never details. Thus, a wheel with spokes was described as a circle or a diamond. Concentric circles were described as one circle or a "smudge within a circle." At lower speeds, she recognized one object at a time. When there was more than one object in the field and she happened to recognize one of them, she selected the one in the left lower inferior quadrant. When colored objects were exposed, she sometimes recognized one of them, usually the one which was present in the left lower quadrant of the projected field.

Visual After-Imagery: The patient claimed that she did not see an after-image after strong light stimulation. There was no flight of colors. The light

of a 40 watt lamp held for five seconds 1 foot (30 cm.) from the eye produced no report of visual after-imagery. However, when the patient was asked to read a short, simple word printed on a white sheet of paper, she was unable to do so for several seconds after light stimulation. Tested two minutes later, she succeeded in reading the word. This occurred on repeated occasions. This transient amblyopia following light stimulation suggested that some of the usual reversible physiologic changes which follow photic stimulation had occurred, although the patient denied having anything like an after-image.

Mimicry: She was able to imitate a simple movement or attitude which required a single performance of one limb or of the face. Her response, however, was a mirror image of the examiner's movements. With more complex acts she was unsuccessful. She could not imitate the acts of closing the eyes and opening the mouth. Most tests of mimicry showed that she was able to execute one performance at a time, but not more than one.

Intellectual Functions.—In general, her memory was extremely poor for recent, and to a lesser extent for remote, events. She could repeat five figures, but she could not repeat five different words. Her visual retention was extremely poor.[23] This defect was more pronounced if there was more than one figure in the presented image. Calculation and spelling were defective. Simple words, such as "cat" or "dog," were spelled correctly. However, she was unable to spell these words backward.

In all these tests a prominent feature was the patient's inability to perform unless there was unity of idea or action. If there was more than one salient aspect of a situation, she was unable to accomplish her task. When instructed to pass two different objects to two people, she did so; but she became confused when instructed to distribute three objects in a certain order to two people. As already noted, she was able to carry out simple commands, such as closing the eyes, opening the mouth or raising her hands; but as soon as the act involved two different and asymmetric parts of her body, she was unsuccessful.

Characteristic features of all her performances were pronounced perseveration and fluctuation. The perseveration was found in her sensory and motor performance and in her speech. The repetition of the acts was sometimes so pronounced that it was difficult to evaluate the particular responses studied. On one occasion she calculated correctly and spelled words which she could not at other times. However, she was unable to perform when the task required her to assume a complex "set." She also showed a general desire to avoid situations which might possibly lead to failure. After having read two or three words, she would stop and remark that she "could not see." With prodding, she resumed her task but soon stopped. There was no such hesitation or fluctuation in performance when she was required to read single small words, each on a separate card. There were also fluctuations in her affect. From time to time the patient burst into tears without apparent provocation; on closer analysis, however, it was found that the crying occurred most often when the patient was confronted with what seemed to her an insoluble problem.

Psychometric and Rorschach Tests: On the Wechsler-Bellevue Intelligence Scale her functioning was comparable to that of a mentally defective patient and was interpreted as indicative of severe cerebral damage with deterioration. Her composite intelligence quotient was 55, with a verbal score of 65 and a performance score of less than 50. Results of the vocabulary subtests led one to infer that her premorbid intelligence had been at least dull normal. She showed complete

23. Benton, A. L.: A Visual Retention Test for Clinical Use, Arch. Neurol. & Psychiat. **54**:212 (Sept.) 1945.

incapacitation in manipulating materials and in perceiving spatial relations. The organic impairment was aggravated by a tendency to perseveration in responses and was manifested specifically in her ability to repeat six digits forward but only two backward. On the Rorschach examination, six of Piotrowski's ten classic signs of cerebral damage were noted. There were only eleven responses for the entire series of ink blots. There were no movement responses, fewer than 25 per cent popular responses, a high percentage of inaccurate forms, repetition of concepts without regard to form and a tendency to automatic phrases at the end of each card. In addition, she was unable to locate or to indicate any parts of the concepts on the plates. Nine of her responses were a perseveration of "kidney." A clue to her mental content was obtained when she associated to "kidney," stating: "Keep you alive.....I had trouble with kidney.....bladder..... I had a miscarriage.....I am still wetting."

She gave only two responses which were of acceptable form quality; however, these two responses were those that are most frequent among the responses commonly given to these cases (popular responses). For this reason, it was assumed that her basic personality structure was not pathologic prior to her present mental illness.

COMMENT

In general, this patient showed the clinical picture of so-called mental deterioration, of the type usually found in patients with disease of both frontal lobes. There was no evidence of aphasia. Routine neurologic examination showed slight sensory disturbances on the right side. However, with special tests it was possible to demonstrate severe disturbances in all modalities on this side. The final clinical diagnosis was degenerative disease of the brain.[23a]

The most prominent features in the patient's case were (*a*) her failure to report one (the one on the right) of two stimuli presented simultaneously on the two sides of the body; (*b*) her inability to accomplish a task which involved two or more aspects of a situation or required a complex "set," and (*c*) the unilaterality in her responses, namely, her consistent "preference" for her left side in perceptual reactions and motor performance. In all her sensory reactions and motor performances there seemed to be a canalization of all available energy into one direction [24] (viz., from her right to her left side). It appeared as though the sum

23a. Since this paper was written, the patient has died. Autopsy showed gross atrophy of the brain, especially in the parietal regions. Preliminary histologic studies made at Kings Park State Hospital showed thickening of the meninges, cortical atrophy, diffuse aggregations of inflammatory cells and a few scattered senile plaques.

24. Goldstein (Goldstein, K., in discussion on Bender, M. B., and Furlow, L. T.: Phenomenon of Visual Extinction and Binocular Rivalry Mechanism, Tr. Am. Neurol. A. **70**:89, 1944; Aftereffects of Brain Injuries in War: Their Evaluation and Treatment, New York, Grune & Stratton, Inc., 1942) is of the opinion that the nerve energy which is available to the organism is a constant. In effecting a performance under pathologic conditions, the "available nerve energy" is utilized better and to a greater extent by the normal cortex, so that an insufficient quantity is left for the use of the diseased cortex.

of the patient's reactions was "ordered," as Goldstein expressed it, toward her left side. On the right side of the body the patient's difficulties in localizing visual, auditory and cutaneous stimuli were extreme.

The defects in localization, the allesthesias and the instances of extinction might have a common physiologic basis. All these phenomena show that the reactions of the organism depend not on excitation of single receptor fields but on total patterns of excitation. The clinical observations described in this paper are therefore difficult to explain in terms of the classic neuronal theory.

There are experimental data in recent neurophysiology which might help in clarifying some of the symptoms in our case. So far, the known neurophysiologic facts seem to bear only on the phenomenon of extinction. Dusser de Barenne and McCulloch found that a decrease in action potentials over the entire sensory projection area can be produced by electrical simulation of certain regions within the sensory cortex.[25] The authors proposed to call these regions sensory suppressor areas. More recently, Gellhorn and Barker[26] extended these studies by activating these suppressor regions by peripheral sensory stimulation. As a result of this peripheral stimulation, the action potentials in the sensory projection area are diminished. Moreover, the suppression of cortical activity occurred in the ipsilateral and the contralateral cortex. The effect was greater in the ipsilateral cortex, and increased with increase in intensity of peripheral stimulation. However, the spread of cortical suppression in these experiments is slow, while extinction in the clinical sense occurs much more rapidly. More studies on cortical suppression are necessary before a neurophysiologic explanation of extinction will be acceptable.

There is even less evidence that the other symptoms described in our case (allesthesia and spatial disorientation) could be reproduced in the laboratory. Dusser de Barenne[27] and Mott[28] each found that hemisection of the spinal cord in the experimental animal produced an effect which they interpreted as "allesthesia." The animal responded to stimulation of the anesthetic side by movements of the extremities which suggested that it felt the stimulus on the normal (unstimulated) side.

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25. Dusser de Barenne, J. G., and McCulloch, W. S.: Factors for Facilitation and Extinction in the Central Nervous System, J. Neurophysiol. **2**:319, 1939.

26. Gellhorn, E.: Effect of Afferent Impulses on Cortical Suppressor Areas, J. Neurophysiol. **10**:125, 1947. Barker, S. H., and Gellhorn, E.: Influence of Suppressor Areas on Afferent impulses, ibid. **10**:133, 1947.

27. Dusser de Barenne, J. G.: Zur Kenntnis der Alloästhesie, Monatschr. f. Psychiat. u. Neurol. **34**:523, 1913.

28. Mott, F. W.: Results of Hemisection of the Spinal Cord in Monkeys, Phil. Tr. Roy. Soc., London **1**:183, 1893.

Similar observations were made on man by Ray and Wolff [29] and Holbrook and de Guitérrez-Mahoney.[30] In the case reported by the latter authors, the pain induced by pinching an analgesic region was localized to the opposite side of the body. However, the patient felt the touch at the point where he was pinched. Thus, one stimulus (pinching) evoked two sensations: touch at the point of contact and disagreeable pain at the corresponding point on the opposite side of the body. Such a response is not the same type of "allesthesia" as that defined by us in footnote 16. This type of double sensation (touch and pain) we prefer to call synesthesia. It is doubtful, therefore, whether we could use the neuronal theories (spread of excitatory process within the neuraxis) of Dusser de Barenne and others to explain the type of allesthesia found in our patients, even though it might explain the type of "allesthesia" produced by section of the spinal cord. As for the disorientation in space, it is impossible to explain this disorder in terms of present neurophysiologic concepts. We are not aware of physiologic data which would demonstrate that the massive disorientation in space shown by our patient could be reproduced by experiment. We must turn to other theories. Instead of assuming that function is based merely on chains of events in separate neural pathways, we might consider the role that "masses of excitation" play in the function of the nervous system, as proposed by Lashley. The phenomena of extinction and allesthesia suggest that the organism's reactions vary with the patterns of excitation. Double stimulation leads to systematically different results than single stimuli. Then there is Goldstein's holistic theory,[2] which interprets behavior as a function of the total substrate rather than of its parts. The symptoms shown in the present case illustrate many of the principles enunciated by Goldstein.

Some of the Gestalt psychologists have further specified the holistic theory by assuming that directed field forces or vectors operate in the substrate (Koffka,[5b] Lewin [31] and Orbison [32]). The common denominator of these disorders is an apparent imbalance between the forces on the right and the left side. The disease process in our patient seemed

29. Ray, B. S., and Wolff, H. G.: Studies on Pain: Spread of Pain; Evidence of One Site of Spread Within the Neuraxis of Effects of Painful Stimulation, Arch. Neurol. & Psychiat. **53**:257 (April) 1945.

30. Holbrook, T. J., and de Guitérrez-Mahoney, C. G.: Diffusion of Painful Stimuli over Segmental, Infrasegmental and Suprasegmental Levels of the Spinal Cord, Federation Proc. **6**:131, 1947.

31. Lewin, K.: Principles of Topological Psychology, translated by F. Heider and G. M. Heider, New York, McGraw-Hill Book Company, Inc., 1935.

32. Orbison, W. D.: Am. J. Psychol. **52**:31-45, 1945. It should be noted that both Goldstein and Lashley criticize the vector theory. Lashley, for instance, says that "its application to specific problems frequently involves specific ad hoc assumption, and its explanations turn out to be only elaborate tautologies."

to have established an abnormal gradient in sensibility largely between the two sides.[33]

Other symptoms recorded in this case might be explained by the vector theory. In another communication it was pointed out that certain forms of disorientation in visual space could be interpreted in terms of an imbalanced interaction between distal hypothetic "forces" (i. e., those deriving from the particular stimulus configurations in the field of vision) and proximal "forces" (i. e., hypothetic background activity in the "cortical field").[10]

In patients with cerebral lesions, permanent changes in the spatial organization of perceptions may be similarly conceptualized as a disequilibrium in the cortical field forces. Defects in sensory pathways, or even in regions outside the sensory pathways proper, lead to a reorganization and redistribution of the remaining forces, which determine the new field of the particular sensation considered. The new field, however, is no longer as stable as in the normal state. For example, in a patient with defective visual substrate certain changes in the stimulus configurations present in visual space will produce a disorientation, although such alterations would not do so in the normal condition. If the lesion is severe enough, the disorientation in visual space may be persistent and not merely incidental to certain conditions in the field of vision. This was the case in our patient. On studying the patient's behavior, it was quite evident that her visual and motor performance in tests for spatial orientation had changed in a systematic fashion, as though they were determined by certain directional "forces." Although we have no definite means of measuring these forces, we could, nevertheless, use them in predicting the perceptual and motor reactions of the patient in other acts.

At present the conceptual framework into which most of our patient's symptoms might be fitted is Goldstein's holistic theory. From this point of view, even the mental symptoms might be considered as an integral part of the systematic alteration in function.[34] The patient's inability to maintain a complex mental set was similar to her perceptual difficulties on double stimulation.

In conclusion, it would seem that our case does not offer crucial evidence for any specific theory; in spite of this, however, a consideration of the various theories prompts us to use methods of study which the ordinary examination does not include. Consideration of various theories

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33. The most conspicuous gradient was between the two sides, but a gradient in sensibility was also present between the proximal and distal portions of the right side extremities. The topographic pattern of sensory dysfunction in the affected extremities was of the type usually found in lesions of the cerebral sensory pathways.

34. Goldstein, K., and Katz, S. E.: The Psychopathology of Pick's Disease. Arch. Neurol. & Psychiat. **38**:473 (Sept.) 1937.

leads to more thorough investigation of the changes in function. For instance, the holistic principle stresses that while a particular function is tested in one region of the body, numerous simultaneous (uncontrolled) changes occur in other parts of the body. Obviously, the changes in other regions are not apparent unless they are tested simultaneously; the method of simultaneous double stimulation, as employed in the study of our patient, has yielded information which routine neurologic examination failed to do. Also, study of the patterns which these dysfunctions possessed showed: (*a*) similarity in alteration of motor and sensory functions (both functions were apparently weakened on her right side, or dominant on her left side) and (*b*) similarity between her sensory and her intellectual performance (in both there was inability to carry out a task which involved simultaneity of performance).

SUMMARY

In a patient with severe mental deterioration, routine neurologic examination disclosed minimal signs of a focal lesion in the brain. Special methods, however, such as bilateral simultaneous stimulations, revealed that the patient perceived best on the left side. Although she felt a stimulus applied to her right side, this percept became extinct as soon as another stimulus was applied to the left side of her body (extinction phenomenon). In these circumstances she perceived only the one on the left side. The extinction phenomenon was found to be present when her cutaneous, kinesthetic, visual or auditory perceptions were thus tested. The patient also showed the phenomenon of allesthesia. Not infrequently she localized an object she saw, heard or felt to the left side, even though the stimulus object was applied to the right side of her midline.

Related to the sensory dominance of the left over the right side was the patient's preferred use of the extremities on the left side and her preference to turn toward her left side. Akin to the allesthesia was the finding of a disorientation in her cutaneous, visual or auditory space on the right side of her body.

Besides the perceptual and motor disorders, the patient showed pronounced intellectual changes. Most conspicuous was the patient's inability to perform a task unless there was unity of idea or action. If there was more than one salient aspect in a situation, she failed in her task.

Theoretic explanations for the phenomena of extinction, allesthesia and spatial disorientation are offered, and the relation between the systematic perceptual-motor disorders and the systematic intellectual changes is discussed. These alterations in function can be best understood when considered in terms of collective action within the nervous system as a whole.

PALLHYPESTHESIA

Depression of the Appreciation of Vibration in Trauma and in Disease; a Preliminary Report

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DETERMINATION of vibratory appreciation by application of the stem of a tuning fork to the bony prominences of the body is part of the routine neurologic examination.

The anatomic pathways for vibratory conduction are described as running from receptor endings in the tendons and muscles through the deep nerves to the posterior roots, to the posterior columns of the ipsilateral side to the nuclei of Goll and Burdach, then across to the median fillet and up to the thalamus and fanwise to the postcentral cortex. Wechsler [1] stated:

> Vibratory sensation is conveyed up to the thalamus practically by the same tracts as joint sensibility (both representing deep or proprioceptive sensation) and, except for cortical lesions, is lost at the same time with it. Only occasionally does one observe dissociation between them. Both have the same significance in peripheral nerve disease, tabes, and other cord affections. It is not sound but a vibratory thrill ("like electricity") that is preceived.

The statement inferring that dissociation of vibratory appreciation and proprioceptive sensibility of the joint, or position sense, is infrequently observed has been seriously questioned by some neurologists, and it will be shown in this study that such dissociation is a common phenomenon.

Gasser and others [2] have shown that vibratory appreciation, light touch sensibility and position sense are carried by the large myelinated fibers (A and B) and that the velocity of the impulse is rapid. They expressed the belief that there is some evidence to show that these fibers are affected earliest in tabes and peripheral neuropathy.

Gross decreases in vibratory conduction have long been recognized in association with traumatic, neoplastic, infectious and nutritional diseases involving the pathways concerned with conduction of vibratory impulses. General impairment of pallesthesia is known to occur in cases of arteriosclerosis of the cord, combined sclerosis associated with

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1. Wechsler, I. S.: A Textbook of Clinical Neurology, ed. 5, Philadelphia, W. B. Saunders Company, 1943, p. 43.

2. Gasser and others, cited by Wechsler,[1] p. 50.

pernicious anemia, multiple sclerosis, tabes dorsalis, polyneuritis and traumatic and neoplastic lesions high in the spinal cord. Lesions of the optic thalamus give rise to impaired vibratory appreciation on the contralateral side.

Roth,[3] in 1941, devised a modified tuning fork designed to quantitate accurately levels of pallesthesia. This instrument, which he called a neurometer, is constructed to deliver 70 decibels of energy at 128 double vibrations per second with a standard decaying time. A thin metal rod is inserted through the stem of the fork to act as a cross bar for the suspension of the instrument from the fingers. For mensuration of duration of vibratory perception at sites other than the fingers, the stem of the neurometer is used in the same manner as the ordinary tuning fork.

Using the neurometer, Roth was enabled to detect small fluctuations in levels of pallesthesia, which could be recorded as percentages of a predetermined standard. He described the normal time of vibration appreciation as 35 seconds ± 5 seconds for the fingers. For the toes, the average normal values were found to be 20 seconds ± 2 seconds. Any values below 30 seconds for the fingers or 18 seconds for the toes were considered abnormal and required explanation in terms of disease. He noted that the results of testing for the vibratory sense in the aged frequently fell to the pathologic levels even in the absence of specific detectable disease of the nervous system.

Roth found decreased values for pallesthesia in approximately one-half the patients studied who gave a history of unconsciousness of variable duration following trauma to the head. In all patients examined who gave a history of unconsciousness of variable duration with simultaneous bleeding from the nose, eyes, mouth or ears after exposure to severe blast he found a grave loss of vibratory appreciation. He was able to detect these impairments many years after the initial injury, and he concluded that pallhypesthesia due to trauma, once established, is irreversible.

Roth found that vibratory appreciation was invariably intact in patients with psychoneuroses of the combat fatigue type. He suggested that this finding be used as a specific differential factor to distinguish patients with psychoneuroses from those with organic injury to the brain resulting from mechanical trauma to the head or exposure to severe blast.

CLINICAL INVESTIGATIONS

Determinations of levels of pallesthesia on 200 patients from the sections of dermatology and venereal diseases of an army general hospital in England gave certain novel and unexpected findings.

3. Roth, A.: Measurement of Vibration Sense: Method and Means, War Med. **4**:280 (Sept.) 1943.

The technic of testing was as follows:

1. The patient is comfortably seated in a quiet room.

2. One hand is presented, palmar surface up and the middle and index fingers separated in the same plane without strain.

3. The neurometer is activated by striking it against the examiner's hand so that a distinct clicking sound is heard, indicating maximum activation of the fork.

4. Simultaneously a concealed stopwatch is started.

5. The cross bar of the vibrating neurometer is then placed on the tips of the presenting index and middle fingers, and the patient is instructed to signify the exact moment when vibratory appreciation has completely disappeared.

The chief difficulty in the quantitative mensuration of any part of the sensorium arises from the variations in intelligence, attentiveness and cooperation of the patient. In order to compensate, at least in part, for this lack of an objective method of testing the verity of the response, the test was carried out at least three times on each hand or other test site at each examination. Patients were tested daily while hospitalized and at weekly intervals thereafter.

Investigations were initially carried out on the fingers. If a difference in the time of vibratory appreciation of more than 5 seconds between the two hands was noted, or if the results for the fingers were within the pathologic range, examinations were carried out on the great toe, malleolus, tibia, knee, iliac crest, elbow and mastoid and for hearing.

It was found that in conditions producing general impairment of vibratory appreciation the factor or percentage of decrease determined for the fingers was generally constant for the rest of the body. With conditions in which local neurologic lesions were suspected or proved a wide divergence was noted in the values at various sites.

Tests on cold hands and feet, as in patients with neurocirculatory asthenia or exposure to external atmosphere, gave unsatisfactory results. Washing the hands in hot water was found to be the best method of overcoming this difficulty. This procedure in no way influenced the results for patients with previously determined low vibratory appreciation due to disease with the exception of a patient with Raynaud's syndrome, for whom very low vibratory ratings were found when the fingers were tested during spasm. The application of heat relieved the spasm, and tests when no obvious spasm was present revealed normal values for pallesthesia.

Investigations were carried out on members of the armed forces and included representatives of both sexes and of the Negro and white races. The ages varied from 18 to 45 years, the majority of persons being within the age groups of 25 to 35 years.

Patients with pyogenic and mycotic infections, parasitic infections and hypertrophies of the skin, as well as those with psoriasis, nummular eczema and neurodermatitis, acute and chronic contact dermatoses, urticaria, hay fever and gonorrhea, gave values for vibratory appreciation within the normal limits as established by Roth. No patient with chancroid or granuloma inguinale was observed during the course

of this study. Levels of from 32 to 28 seconds appear to be of borderline nature rather than low normal, since they apparently represent some slight or recovering neuropathologic process, as well as occurring in apparently normal persons. The number of patients in each group and the distribution curve for pallesthesia values are shown in the table and in chart 1.

DEPRESSION OF VIBRATORY APPRECIATION

Depressions of vibratory appreciation are considered with reference to the disease apparently responsible for the deviation.

Seventy-five per cent of the men with a history of unconsciousness of variable duration following trauma to the head showed pronounced

Values for Pallesthesia in Trauma and Disease

	No. of Cases	Vibratory Perception Time for Fingers, Sec.*												
		40	35	30	28	26	24	22	20	18	16	14	12	10
Normal	49	10	60	30	..	..	..	..	..	..	..	..	..	..
Traumatic injury to brain														
Mechanical	24	..	12	8	8	8	20	12	8	8	12	..	..	..
Blast exposure	10	..	..	..	..	10	50	20	20	..	..	..	..	..
Hypothyroidism														
Untreated	21	..	..	..	14	14	14	28	7	14	7	..	..	..
Treated	15	21	35	21	7	7	7	7	..	..	..	..	..	..
Lymphogranuloma venereum	5	..	..	..	..	..	60	40	..	..	..	..	..	..
Syphilis														
Early infectious	26	4	4	..	..	16	16	12	16	8	4	4	8	8
Latent	20	..	5	..	..	20	20	15	25	5	5	..	..	5
Late active	7	14	..	..	..	14	28	..	..	14	14	14	..	..
Pallhypesthesia, unclassified	23	..	..	..	16	36	28	4	4	4	..	..	..	..

* The number of cases for each value are expressed as a percentage.

diminution of the time of perception of vibration. The injury had occurred ten to twenty years previously in the majority of these patients, lending credence to Roth's belief that the depression accompanying pallesthesia in patients with head injury is a permanent condition. Whether the patients in this small series who gave normal values for vibratory appreciation at the time of testing had ever had demonstrable pallhypesthesia in the past is unknown.

Depressed vibratory appreciation was found in all patients who gave a history of unconsciousness with bleeding from the mouth, nose or ears after exposure to severe blast. All injuries were recent, and reexamination during a short period of observation revealed no significant fluctuations.

Patients with untreated, mild to moderate hypothyroidism showed pronounced pallhypesthesia. Some patients under active treatment with

thyroid gave borderline values at the time of first testing with the neurometer. When the dose of thyroid was continued or increased, some of the patients showed an elevation of the level of pallesthesia to the average normal value. The following case is representative of the reactions of patients of this type.

A white man aged 34 had psoriasis of four years' duration; the disease was aggravated during his stay in England. While he was under treatment for this condition, the diagnosis of concomitant moderate hypothyroidism was made and was confirmed by a basal metabolic rate of —23 per cent and a cholesterol content of the blood of 300 mg. per hundred cubic centimeters. Treatment for the hypothyroidism was irregular, and a relapse of the psoriasis led to his admission to

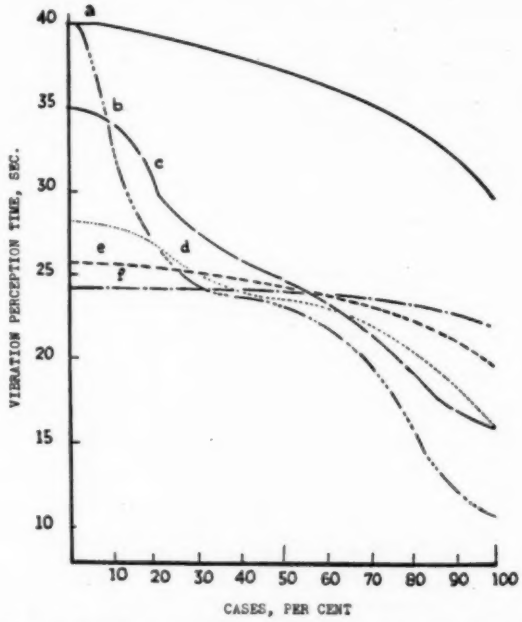

Chart 1.—Pallhypesthesia in cases of trauma and disease. In this graph, *a* indicates values for patients with normal vibratory appreciation; *b*, values for patients with syphilis; *c*, values for patients with mechanical injury to the brain; *d*, values for patients with hypothyroidism; *e*, values for patients with blast injury to the brain, and *f*, values for patients with lymphogranuloma venereum.

the hospital, at which time pallesthesia was noted for 30 seconds on both hands. With the regular administration of thyroid U. S. P., 64 mg. three times a day for four weeks, striking improvement was noted in the signs and symptoms due to the hypothyroidism. A gradual increase in time of vibratory appreciation was noted on repeated testing. The average weekly ratings were 35, 37, 41 and 41 seconds, respectively, for both hands. No deviation in position sense was noted at any time in this patient.

A small group of patients with grave depression of pallesthesia failed to show any response to thyroid during a period of treatment of three weeks. Whether continued treatment or increased dosage

will result in a return of the vibratory perception to normal values cannot be predicted.

Routine investigation of syphilitic patients in the early infectious, latent and late active phases of the disease revealed severe depressions of pallesthesia in almost all instances. Pallhypesthesia was noted when the primary lesion was less than one week old and in patients whose syphilis had apparently been completely quiescent for from twelve to twenty years after treatment. No abnormalities of position sense were noted in any of these patients. Depressions of vibratory appreciation were found in cases of asymptomatic, as well as of symptomatic, neurosyphilis of all types. In the patients with asymptomatic neurosyphilis tests of position sense gave normal values in every respect.

Diminution of vibratory appreciation remained constant on daily testing prior to institution of treatment. The withdrawal of 15 cc. of spinal fluid before beginning antisyphilitic therapy produced no significant changes.

A sharp fluctuation in vibratory appreciation occurred in some patients under treatment with penicillin (40,000 units every three hours for a total of sixty doses) which might be interpreted as a Herxheimer reaction. This deviation was followed by a gradual rise in the level of pallesthesia, which, however, did not at any time attain normal rating.

The following case is representative of the findings in cases of primary, secondary and latent syphilis.

A Negro aged 22, with a history of exposure to syphilis five weeks previously, presented a mild inflammation of the left inguinal lymph nodes and two shallow ulcerations on the dorsal shaft of the penis, from which Treponema pallidum was recovered in large numbers. Except for grave pallhypesthesia, the physical examination, including neurologic investigation, revealed nothing significant. No abnormalities of position sense were elicited.

Serologic tests of the blood, studies of the spinal fluid, urinalysis and roentgenologic examination of the heart and great vessels were reported to reveal nothing abnormal. A blood count was within normal limits. The Frei intradermal test gave a 3 plus reaction and the Mantoux test a 2 plus reaction.

Pallesthesia values before treatment were 14 seconds for both hands. After the sixth dose of penicillin, which had produced no local or general reaction, the level of vibratory appreciation of both hands rose to 22 seconds and decreased within the next two days to 14 seconds, persisting at this level with slight fluctuations during the rest of the period of active treatment. Daily investigations during the post-treatment period of observation showed a remarkable similarity in the values for the two hands and a tendency to gradual elevation of the level of vibratory appreciation. Four weeks after completion of treatment these values had risen to 22 seconds for both hands.

Herxheimer reactions, with local manifestations, accompanied with increase in temperature, which formerly occurred in 80 per cent of syphilitic patients after treatment with penicillin, had not been observed

in this hospital since January 1945. At that time a change in the color and the solubility of the penicillin used was noted. However, the sudden rise in the level of pallesthesia, with return to the previously established low levels, occurred in 3 cases of primary syphilis within the period in which the Herxheimer reaction would normally be expected to occur (with the previous types of penicillin available), and this finding may represent the only residual reaction of the suppressed phenomenon (chart 2).

A young Negro with adequately treated latent syphilis, neurocirculatory asthenia and phlyctenular keratitis gave normal values for vibratory perception.

A white woman aged 22 with secondary syphilis showed moderately low values for pallesthesia during the active phase of the disease and normal average values for both hands two weeks after penicillin therapy.

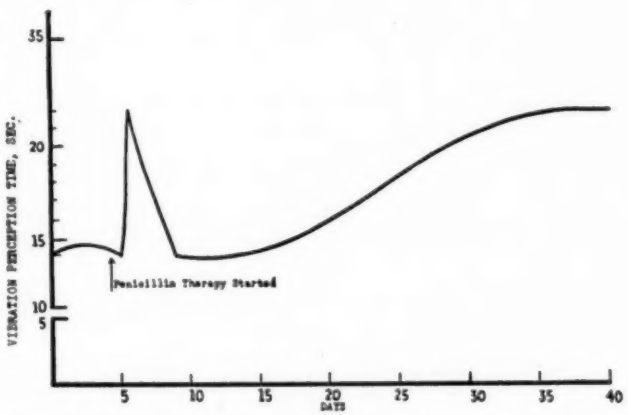

Chart 2.—Herxheimer type of reaction in levels of pallesthesia occurring during treatment for primary syphilis.

A white man aged 35 with primary syphilis showed a 50 per cent reduction of pallesthesia before and during penicillin therapy and an average normal rating for both hands immediately after completion of the treatment.

A Negro with symptomatic, diffuse meningovascular syphilis presented low normal levels of vibratory appreciation previous to therapy. Repeated daily examinations during and after penicillin therapy revealed a further depression of the pallesthesia levels to the pathologic category.

A white man aged 20 with a persistently negative serologic reaction of the blood for syphilis had a very low level of pallesthesia. Further study of this patient revealed the typical stigmas of congenital syphilis with symptomatic involvement of the central nervous system.

Five Negro patients with uncomplicated acute or chronic lymphogranuloma venereum showed a striking diminution of vibratory appreciation. Daily tests while they were under treatment with sulfonamide drugs revealed no significant changes. The short post-treatment period of observation was insufficient to show whether eventual recovery will occur. The small number of patients with uncomplicated lymphogranuloma venereum observed in this series does not permit any assumption as to the actual incidence of pallhypesthesia associated with this disease.

The already lowered values for the vibratory sense noted in association with syphilis were apparently not further lowered by the acquisition of lymphogranuloma venereum, as was demonstrated by a patient who acquired acute, severe lymphogranuloma venereum during the post-treatment period of observation following penicillin therapy for primary syphilis. It is possible that the frequent occurrence of lymphogranuloma venereum in Negro patients with syphilis may tend to keep statically depressed pallesthesia values which might otherwise rise to more nearly normal levels as a result of antisyphilitic therapy.

COMMENT

Pallhypesthesia was found in patients with a history of unconsciousness following trauma to the head and after exposure to severe blast which produced unconsciousness and bleeding from the eyes, ears, nose and mouth, in accordance with observations previously reported by Roth. In spite of the small number of cases studied, the fact that the patients investigated were unselected and that no exceptions were found among the patients with injury to the brain following exposure to severe blast indicated that the incidence of pallhypesthesia in these two traumatic conditions is probably high.

Significant depression of vibratory appreciation in association with hypothyroidism and with lymphogranuloma venereum and all phases of syphilis other than far advanced lesions of the spinal cord have been hitherto unsuspected and are described here for the first time.

Diminution of normal thyroid secretion appears to produce significant pallhypesthesia. The finding of a concomitant 30 per cent reduction of vibratory appreciation in a patient with mild hyperthyroidism may indicate that patients with toxic goiter may have simultaneously a decreased secretion of normal thyroxin.

An interesting point in the entire study is concerned with the apparent specific involvement of the fibers carrying vibratory impulses without affecting the other proprioceptive fibers supposed to be closely associated anatomically and previously usually considered to be almost always simultaneously injured.

The absence of clinical findings except for pallhypesthesia in the cases of early and latent syphilis may mean that the fibers carrying vibratory impulses are the most labile and sensitive of the entire nervous system. Changes in position sense were not found in any of these cases.

On the other hand, the elevation of vibratory appreciation previously depressed in occasional cases of early pernicious anemia and alcoholic polyneuritis, and, as is shown in this paper, in some cases of hypothyroidism under appropriate treatment, indicates that the damage is not necessarily always irreversible. If that is so, the persistently low values found many years after what was considered adequate therapy for syphilis may have clinical significance. Any modification

of treatment which produces a rapid or permanent change of pallhypesthesia to a normal level may be of prognostic importance. The tendency to elevation of vibration appreciation following penicillin therapy, even though the levels do not attain the normal scale, suggests that a modification of this treatment, perhaps increased dosage, might produce greater benefit.

The number of patients treated with various oxophenarsine hydrochloride and bismuth technics who were available for this study was insufficient to permit any final conclusions as to the effectiveness of this medication in raising depressed pallesthesia values. The patients seen had completed treatment from six months to twelve years previously, and levels for all were in the pathologic categories with the 1 exception previously described.

The diminution of vibration appreciation noted in patients with lymphogranuloma venereum is added evidence that this infection produces a generalized disease in which severe involvement of the central nervous system is a frequent manifestation.

Of the groups of patients with pronounced pallhypesthesia uncovered in this study and as yet unclassified as to the etiologic factor involved, some may eventually be classified with the groups already described, or may be found to be representative of other diseases, which have as yet not been considered to have impairment of vibratory perception as one of their manifestations. The exigencies of the service have been responsible for the movement of these patients before studies leading to a proper classification could be completed.

SUMMARY AND CONCLUSIONS

Investigations of vibratory appreciation by means of the Roth neurometer are described in a series of 200 unselected patients in the sections of dermatology and venereal diseases of an army general hospital.

Normal vibration time in the toes and fingers as determined in this series agreed with the established levels utilized by Roth.

The majority of patients with a history of mechanical trauma to the head and all patients with cerebral injury following exposure to severe blast in this series showed a pronounced diminution of vibratory appreciation, in agreement with Roth's previous description.

Depression of pallesthesia in patients with dyscrasias of the thyroid gland is described for the first time. All patients with untreated, mild or moderate hypothyroidism showed a grave depression of vibratory appreciation. Elevation of levels of pallhypesthesia occurred in some patients to normal values after treatment with thyroid.

A grave diminution of pallesthesia was noted in all cases of uncomplicated lymphogranuloma venereum.

All but 3 of 53 patients with syphilis, in all stages, showed a severe impairment of pallesthesia. These exceptions occurred in single cases of primary and latent syphilis and syphilis of the central nervous system, respectively. Treatment with penicillin produced the equivalent of a Herxheimer reaction in the levels of vibratory appreciation of 3 patients with primary syphilis. A gradual rise in the curve for pallhypesthesia was noted in the post-treatment period. This elevation was, with 2 exceptions, never sufficient to attain normal levels. Of the patients treated with oxophenarsine hydrochloride and bismuth schedules, the pallesthesia values were found to be in the pathologic group, even as long as twelve years after termination of therapy.

A group of 23 patients with low vibratory ratings was found. Because of insufficient time for study, these patients were not classified as to the etiologic factors in the depressed levels of vibratory perception.

The small number of patients studied makes all the findings tentative, until larger groups are investigated.

211 Doctor's Building (2).

PSYCHOSIS FOLLOWING LOBOTOMY IN A CASE OF SEXUAL PSYCHOPATHY

Report of a Case

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THE MASS of favorable reports on lobotomy recently prompted Halstead, Carmichael and Bucy [1] to comment critically:

> Unfortunately, aside from their value as vital statistics it is impossible to assess the validity of these findings. At no point have there been other than superficial attempts made *to standardize* the criteria for the pre-operative and post-operative clinical status of the patient. Not a single patient has been adequately studied. For a moral and social responsibility to do this, there has been substituted a phenomenal array of case statistics. Unfortunately, the pyramiding of *unknowns* is scarcely a pathway to knowledge.

This paper is an attempt to remedy the alleged lack in the literature of a single case adequately studied. In 1942, one of us (R. S. B.) collaboratively reported the apparent recovery of a patient with sexual psychopathy after lobotomy.[2] The patient, a sexual offender without psychosis, entered prison in 1939, at the age of 52. To relieve his extreme tension and obsessive-compulsive symptoms, a lobotomy was performed in 1941. The report for publication was made six months after operation, while the patient was still in prison. After his release he came to Chicago, where, five years after operation, he was discovered in the locked ward of the Veterans Administration Facility at Hines, Ill. We established the identity of the patient by letter and exchanged observations. Further studies were made in order to obtain a complete set of comparable data from the two sources.

REPORT OF A CASE

The family history, as well as the material in sections B and C, was obtained from the patient before operation by one of us (R. S. B.) and

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1. Halstead, W. C.; Carmichael, H. T., and Bucy, P. C.: Prefrontal Lobotomy, Am. J. Psychiat. **103**:217 (Sept.) 1946.

2. Banay, R. S., and Davidoff, L.: Apparent Recovery of a Sex Psychopath After Lobotomy, J. Crim. Psychopath. **4**:59 (July) 1942.

after operation by the other (J. W. F.). This is supplemented with data from the patient's prison record and with information from relatives of the patient, obtained by Mrs. Mary Diggles, social worker at Veterans Administration Facility, Hines, Ill.

A. FAMILY HISTORY

The patient's father died in 1929, at the age of 85, after suffering for ten years with asthma, presumably of cardiac origin. A New England traveling salesman, he came home on weekends only. In excellent health prior to the onset of his asthma, he was a steady worker, kindly, reserved and nonassertive. The patient described him as "a quiet person. He used to bring me presents when he came home. He was gentle and never beat me." At the age of 35 he married a socially prominent woman of the same town, eight years his junior.

The mother died in 1934, at the age of 82, of cerebral hemorrhage. In contrast to her husband, she was obese, worrisome, insomniac, domineering and excessively devoted to the patient. The patient (hereafter referred to as J. S.) recalled with tenderness the times he sang in the church choir with her or accompanied her on the piano when she sang at social events. He described her as "very kind-hearted. She would give me anything I wanted. Of course, when I did wrong, I would receive a beating on the rear end, about two or three times a week." These beatings, invariably followed by a compensatory kiss, were administered on his bare buttocks until he was 16 years old.

While his father was nominally a Unitarian, of eighth grade education, his mother was a staunch Methodist, with a high school background. The parents never displayed affection openly and slept in separate beds. When the father complained of her extravagance, the mother, who received rent from a house she owned independently, paid no attention. Likewise, his protests against her addiction to spanking J. S. were in vain. Despite these differences, the marriage endured.

J.S. was the youngest of 3 siblings. The oldest, a girl, died of diphtheria at the age of 6, before J. S. was born. A brother, eight years older than he, died of peptic ulcer at the age of 56. The brother, who resembled the father and was preferred by him, had 3 children who are well-to-do, highly respected members of the community. The maternal grandfather died at the age of 93, shortly after admission to a state hospital with a diagnosis of psychosis with cerebral arteriosclerosis. He had been a good provider and was happily married. His wife died at the age of 94. A maternal uncle committed suicide while ill with peritonitis. No data on the paternal relatives were available.

B. EARLY DEVELOPMENTAL, EDUCATIONAL AND SOCIAL HISTORY

J. S. was born in 1887 in a New England town of 15,000, where he lived with his parents until he was 42 (1929). He was enuretic until the age of 15. In general he was a "good" child but occasionally displayed temper tantrums to his mother, whom he "worshiped." He considered his childhood happy.

He completed two years of high school and a year of secretarial school at the age of 18. He did well in all subjects except mathematics. In school he was never punished for any delinquency. While he had many friends, he characterized himself as "shy and retiring" and "a stutterer." He attended the school dances but never learned to dance. Bookishly inclined, he fancied himself a dentist or a doctor, but made no actual step toward becoming either.

As an adult he lost his shyness but retained a slight stammer. Relatives considered him an "intelligent, happy-go-lucky person with a wonderful personality." Since he played the piano and sang well, he earned social popularity. He dressed meticulously and took pride in his collection of books, especially the "Harvard Classics," and later his musical recordings—in partticular, Gounod, Brahms and Mozart. Things never seemed to bother him, a fact which his niece attributed to the solicitude of his mother, who selected his clothes as long as he lived with her. He was active in boys' groups as a scout master and a choir teacher. In 1929, when forced to leave town because of his homosexual activity, he appeared casual as he made preparations for departure. From New York he continued to send money to his mother, but often asked her to return it. This she did, sometimes adding money of her own. Called home in 1934 with the news that she was dying, he left her deathbed, to be found several hours later on one of the street corners, talking with a group of young boys.

Drafted in July 1918, he served six months in the Army, with two promotions. There was no history of court-martial, hospitalization or overseas duty. He smoked but did not drink, gamble or use narcotics. At home he attended the Methodist Church regularly with his mother, but after her death he was converted to Catholicism through a homosexual friend whom he met in a park. Prior to the age of 52 he had never been arrested.

C. PREOPERATIVE DATA

Work History.—Commuting 10 miles (16 kilometers) to Boston during the first five years of his secretarial career (1906-1911), J. S. worked under five different employers. He denied being fired, attributing these changes to his "nervousness." For eighteen years (until 1929), he earned $35 a week as secretary to the superintendent of schools in his home town. Forced to leave town because of his homosexuality, he went to New York city. During the next five years he earned $43 a week as a stenographer for a bankers' association but lost his job because a new employer preferred women stenographers. During the following year and a half (1934-1936) he had no steady employment and required "relief" for six weeks. Until he was sentenced to prison, three years later, he worked for several WPA projects as a stenographer at $25 a week.

Psychosexual History.—J. S. played with dolls until he was 7, delighted to dress as a girl at 8, and periodically slept with his mother until he was 10 years of age. When he was 12, she caught him in mutual sex play with another boy. Indignantly dismissing the other boy, she began spanking her son, whose trousers were already down. His penis remained erect. Thereafter, he managed to make known to her various acts, notably stealing and masturbation, to which she consistently responded with spanking. Further incentive to his provocative behavior was provided by his father, who gave him money for each whipping. When J. S. was 14, the spankings invariably induced ejaculation. Two years later, his mother caught him in passive anal intercourse with a friend. The flagellation that followed was the last such act committed by her. Meanwhile, J. S. had learned to beat himself, using "the same strap mother used," and later induced friends to spank him. This led to homosexual acts, anal and later oral, preferably in the receptive role, without his sacrificing the dominance of his original masochism. In addition, he masturbated almost nightly, with phallic fantasies. Pedophilic as an adult, he became the source of a limited, but regular, income for many small boys. For want of a boy, he would take a man, but never a girl or a woman. Sexual thoughts concerning women were both disgusting and sacrilegious.

Only twice did his sex life lead to serious conflict with society. In 1929 one of his conquests, a Catholic boy of 13, was advised by the priest to tell his father. It was only with the help of his cousin, the mayor, that J. S. escaped with mere expulsion from the town. The second episode occurred ten years later. At a secluded beach, he approached two 7 year old boys. Giving each 25 cents, he

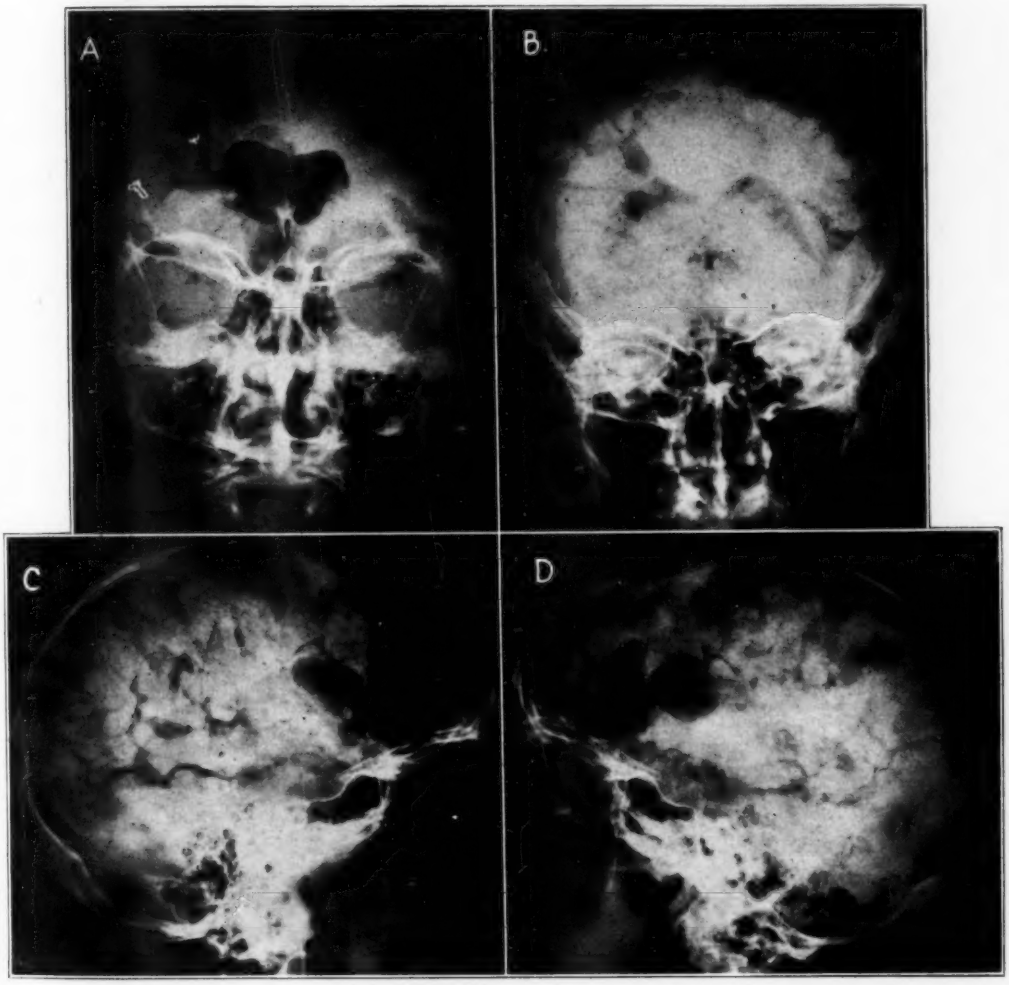

Fig. 1.—*A,* Anteroposterior view, showing excess air in the subarachnoid space over the right frontal area. That none is seen on the left side may be due to an artefact. Both anterior horns are moderately dilated, the right being more enlarged than the left. The third ventricle is slightly dilated.

B, posteroanterior view, showing excessive air in the subarachnoid spaces of both sides. The temporal horns are probably within the limits of normal size. The trephine holes appear to be on each side of the lower end of the coronal suture.

C, right lateral view, showing a general increase in size of the subarachnoid space, being most prominent in the frontal area. The trephine holes tend to obscure it, but the anterior horns of the ventricular system appear enlarged.

D, left lateral view, showing the same changes as in *C,* except that less air appears in the posterior area. The ventricles are poorly outlined.

The interpretation is generalized atrophy of the brain, being greatest in the frontal area.

induced them to undress and go into the water with him. Coming out, he handed each child a homemade cat-o'-nine-tails, which he persuaded them to use on his buttocks. Afterward, fondling their buttocks and genitalia, he had them play with his penis until he ejaculated. Two weeks later, in another park, the children, this time accompanied with their parents, recognized him. On Aug. 25, 1939, J. S. entered Sing Sing Prison to serve a term of two and a half to five years.

Medical History.—Between the ages of 6 and 8 years, the patient suffered from pinworms, which his mother treated twice a week at bedtime by applying "sweet oil" to his anus. At 10 he had scarlet fever, after which he began to stammer. In an attempt to cure his enuresis, he was circumcised at the age of 14. Between the ages of 25 and 35 he had four rectal operations for fissures and fistulas, and at 42, a skinning operation for rectal eczema. He slept poorly and was constipated. The insomnia he often overcame by masturbation; the constipation, by inserting rectal suppositories.

While he considered himself "temperamental," he had no neurotic complaints other than constipation and insomnia until he was about 30 years old. He then began to have headaches, relieved only by sexual gratification, preferably with small boys. At 52, when he was arrested for such gratification, he was sentenced to prison rather than to a psychiatric institution because his abnormal behavior was diagnosed as a sexual psychopathy. After more than a year in prison, he was referred to one of us (R. S. B.) in an agitated state. A year of psychotherapy brought no relief. Lobotomy was recommended because his history justified his fears, his symptoms included tension, depression, obsession and compulsion and he was considered otherwise incorrigible. Following the method of Freeman and Watts, Dr. Leo Davidoff performed the operation with use of local anesthesia on Nov. 22, 1941.

> When the right side of the ventricle was reached, the needle was reinserted slightly more to the anterior. On each side then, the needle was swept downward toward the orbital plate and upward toward the vertex. Afterwards a narrow peri-orbital elevator was inserted on each side and the incision repeated in order to make sure that the pathways were cut.[3]

In the immediate postoperative period, J.S. was confused, disoriented, incontinent and euphoric; he masturbated, fingered his rectum and sucked the feces-laden finger. All these symptoms disappeared by the third postoperative week.

Mental Status.—In October 1940, J.S. was referred to one of us (R. S. B.) because of his extreme nervous tension. He appeared timorous, meek and abjectly apologetic. He stuttered and trembled; his hands perspired, and his voice quavered. At times he strenuously closed his eyes as if to blot out the present exigency. He was filled with self pity. Overwhelmed with fear of committing future sexual assaults on children, he was obsessed with the idea that on release from prison he might commit an act punishable by death. When not in panic, he was preoccupied with masochistic homosexual fantasies, masturbating compulsively two or three times daily. He complained of headaches, insomnia and a sense of weakness but had no delusions, hallucinations or defect in sensorium. Intelligence was bright normal, or even superior. The educational supervisor of the prison, to whom J. S. was assigned as secretary, characterized him as a capable, fast and conscientious worker, handicapped only by his agitated state. His judgment was not impaired, and his insight was fairly good. In prison, at any rate, he was painfully aware of his abnormalities and begged for help, accepting the lobotomy eagerly.

Physical Examination.—Examination in November 1941, shortly before the lobotomy, revealed that J. S. was a thin, well developed, slightly bent man, whose white hair and deeply lined face made him appear somewhat older than

his 54 years. Five feet 9 inches (175 cm.) tall, he weighed 134 pounds (60.8 Kg.). His blood pressure was 110 systolic and 80 diastolic and his pulse rate 76. Except for evidence of vasomotor instability, the physical examination, including neurologic tests, revealed nothing significant. The genitalia and distribution of hair were those of a normal man.

Laboratory and Psychometric Studies.—A complete blood count, urinalysis, roentgenographic examination of the chest, electrocardiographic study and determination of the visual fields, all made within a few days before operation, revealed nothing abnormal. The basal metabolic rate was + 14.5 per cent. The Army Alpha Test (Bregman revision, Form A), administered on the day of his admission, gave him a score of 125, a mental age equivalent to 17 years and 11 months and an intelligence quotient of 119.

D. POSTOPERATIVE DATA

For a year after operation, J. S. remained in prison, under the observation of one of us (R. S. B.). On his discharge, in November 1942, he reported weekly to the parole board until his parole period expired, in May 1944. In prison, he had been befriended by a Pentecostal minister who had made professional visits to Sing Sing Prison, and who once casually invited J. S. to his new home in Chicago. Finding the minister, J.S. then stayed in Chicago.

When J. S. was discovered in the hospital at Hines, his recent memory was so poor that none of his statements concerning his postoperative career could be relied on. The information that constitutes the postoperative data came from independent sources. The first source was the social service reports. These are records of interviews with Chicago employers and friends of J. S. and the record of a previous Chicago hospitalization. The second source was an old trunk belonging to J. S., which was delivered to the hospital. It contained, among other things, a diary and notes written after operation. The third source was the three month hospital record, which includes a diary the patient kept while in the ward. These three sources, together with the parole record and the postoperative observations of each of us, form the basis of the remainder of this report.

Work History.—The parole report gives the work history from the date of the prison discharge, Nov. 10, 1942, until the expiration of parole, eighteen months later. On November 13, J. S. started to work as a porter, quitting one week later because the job was too hard. On November 24, he obtained a stenographic position at $15 a week but quit three weeks later because of the long, irregular hours. When the parole officer visited the employer, he was told that J. S. was "slow and at times made many errors," yet was well liked and would be given better hours. J. S. resumed work for him and continued until the employer left, six months later. His successor was much more difficult to work for. Thereafter, until J.S. obtained a better position, he was frequently absent, losing pay, but not his job. On Aug. 23, 1943, he found a new job as a stenographer at $25 a week. For Christmas, his boss gave him a raise of $2.50 a week and a present of $25. Two weeks later he was fired for inefficiency. On February 10, he found work as a messenger at $25 a week. His new employer, seeing that J.S. found such work arduous, placed him in the clerical department but fired him a week later for inefficiency. On March 13, J.S. took a second job as a messenger at $25 a week. This he apparently retained until he reported to the parole board for the last time, on May 25, 1944.

When J.S. came to Chicago, in June 1944, his friend the minister found him a room and employed him to type and file letters. In October, the minister secured him a job as a "secretary" in a laundry. The proprietor, an official of the Christian Industrial League, conducted services in his laundry for the benefit of his employees. Shortly after hiring him, he recognized that J.S. was confused, unreliable and unable to work. Although he maintained a charitable interest in him and paid the hospital bill when J.S. broke his arm, he would not rehire him. From March to November 1945, J.S. did odd jobs—typing and passing out handbills from door to door. His performance as a handbiller was characterized by his employer, who continued to give him small gifts long after he fired him, as "undependable in every way. Absolutely didn't know what he was doing." In November the minister obtained work for J.S. in a funeral home at $15 a week and lodging. He was required to answer the phone, type and be present at each funeral. Because he would leave his job and was cheerfully incompetent when available, he was fired two weeks later. This was his last known job. Shortly thereafter, he received an inheritance of $1,000 from an aunt.

What happened to the patient and to the $1,000 between December 1945 and February 1946, when he was next heard from, is unknown. J.S. complacently explained that he lost the money when a "drunk" pushed him from a bridge into the Chicago River. From February until September he lived in the Christian Industrial Shelter. Here he was provided with food, room, and $1 a week for laundry and pleasure. He then moved to the Loop Gospel Mission, a similar organization, but was asked to leave six weeks later because he "made mistakes in his clothes [incontinence] and was too lazy to work around the dormitory." He lived next in a rooming house, where he was discovered in squalor, half starved, bedbug-bitten and exuding a profound stench. Obviously incontinent of bladder and bowel, J.S. was separated from his clothes only with the help of soap, water and scissors. The infection of his foot was then noted, and he was brought to the hospital at Hines,

Psychosexual History.—The parole report gave J.S. a clean slate with respect to deviate sexuality. On six occasions J.S. told the parole officer that he was completely free of abnormal desires, and in the final interview he declared himself "physically and mentally free to live a good, clean life."

The diary he wrote in 1943 overlaps the period during which he was on parole. Its reliability is indicated by his accurate recording of all but two of his appointments with the parole officer. The two failures occurred during the twenty-four days in which he made no entry in his diary. Sixty-seven visits to parks were recorded. The frequency of these visits varied with the month, thus: one in January, four each in February and March, six in April, nine in May, seven each in June and July (21 days were blank in these two months), twelve in August (three days were blank), seven in September, six in October, four in November and none in December. Thirteen parks were named, one of them (Prospect Park) twelve times, but the name was often omitted. On four occasions J.S. recorded two parks on the same day. The following dates are given, with their complete entries:

March 14: Fair—mild. Sick with a cold. Home all day. To Prospect Park in evening. Met man.

July 13: Fair—hot. Left 4 p. m. Met boy in park tonight.

August 8: Fair—hot. Went to Church in a. m. To Ft. Green Park in afternoon—sailor. Hard day *No food.*

August 25: Fair—warm. To Central Park tonight. 2 sailors.

October 12: Fair—milder. Holiday. Went to Staten Island. Found a number there.

Besides the diary, the trunk contained clippings from newspapers and magazines, which, from the condition of the paper, appeared to be of recent origin. Seven of these were pictures of boys: 3 of boys smiling; 1 of a boy lying on the ground crying; another of a boy in swimming trunks, and 1 of a boy taking a bath. The last was a sketch of two boys fighting, one on his back and the other on top, lunging a blow. Three cartoons showed a man being whipped. In another cartoon, a woman held a whip in one hand and, in the other, a leash with three small boys attached. Finally, there was a picture of a rattlesnake coiled to strike.

In addition, there were thirty-two scraps of paper bearing the handwriting of J.S., three in green ink and the rest in pencil. Although undated, none of the papers appeared to be old or yellow, and the pencil writing was sharp and distinct. Stamped on one, but penciled out, were the name and Chicago address of the patient's minister friend. The notes were all obscene, with 61 references to a variety of sexual practices, including even coitus. In the order of frequency, the perversions were: masochism (spanking), 21 references; anal intercourse, 12 references; heterosexual relations, 9 references; fellatio, 8 references; voyeurism, 5 references; anilingus, 4 references, and masturbation, 2 references. The two mildest and briefest notes are reproduced in full:

1. Please *FEEL OF MY BARE ASS AND COCK.* I have *no drawers* on. $.25 if you will *PULL* MY *COCK* HARD $.25.

2. Sammy, if we could be *all alone* somewhere and no one would know anything about it if I stripped off *bare* naked, and laid down on the floor or bed would you *spank* my nice soft ass just as *hard* as you could with your belt and *rubber hose.* I haven't had a *spanking* for a long time and how I want one. Perhaps you might know of some place. Would pay you $1.00.

When confronted with these notes, in an attempt to learn why he wrote them, J.S. yielded no reliable information. Because of his suggestibility, he "admitted" alternately that the boys named in the notes were real or imaginary. Although it is probable that he continued to practice perversions in Chicago, the evidence suggests that such episodes were well hidden. Thus, the laundry employer and the heads of the Shelter and Mission were not aware of his homosexuality. At the hospital at Hines, furthermore, there was no record of any homosexual activity. Yet he had firsthand knowledge of the parks and described several homosexual attachments in Chicago, one of which is known to have had a basis in reality. This concerned a boy of 17, who worked at the laundry with J.S. and whose unsavory character led the laundry owner to fire him. J.S. was known to be inexplicably attached to this boy and was heard inviting him to his room. The two often left work together.

Medical History.—After the immediate postoperative confusional state, J.S. emerged amazingly tranquil and apparently free of his erotic fantasies, but still courteous, meek and apparently intellectually intact. The only deficit noted was a reduced energy output. The parole record contained few references to his health or mental status. In December 1942, the parole officer reported, "His memory is very poor . . . ; he gets lost very easily and cannot seem to remember directions." Ten days later, on losing a job, J.S. explained, "My head was getting in bad condition." Later, he again complained of his memory, blaming the operation.

The next reference to his health is in a report from Presbyterian Hospital, Chicago, stating that he was their patient in March 1945. On admission he was found to have a fracture of the left humerus, incurred when he fell on the icy pavement ten days previously. The intern noted: "History unreliable—the patient is confused and has ideas of definite psychotic nature." After the diagnosis of fracture, he added, "Major psychosis—schizophrenia (?)" A staff physician at Presbyterian Hospital informed one of us (J.W.F.) by phone that he remembered J.S. and considered him psychotic.

In the interval between his two Chicago hospitalizations, J.S. was known to complain of headaches; his mental condition can readily be inferred from the postoperative work history. On admission to the hospital at Hines, on Dec. 20, 1946, he presented a swollen and tender left ankle and foot. Placed in the surgical ward with a diagnosis of "severe cellulitis of the left foot and lower leg," he was given penicillin. Two weeks later the infection cleared and he was walking. Because he was confused, irrational and enuretic, a psychiatric consultation was requested. The consultant noted impairment of memory, circumstantiality, poor orientation for time, confabulation and childishness. With a tentative diagnosis of psychosis with cerebral arteriosclerosis or presenile psychosis, the patient was transferred to the psychiatric ward on Jan. 6, 1947.

Mental Status.—At the Hines hospital, J.S. was a gentle, friendly old man, well liked by the staff. Occasionally appearing studious and dignified, he was more often childlike. After the daily ward round meeting, he often beckoned me (J.W.F.) back and cupping his hand over his mouth, announced: "Mr. —— is coming to see me at 4:30. (Pause) He's bringing me something! (long pause, while his eyes fairly gleamed) He's bringing me a bag of candy!!" The same "message," or something equally trivial, was repeated frequently. Superficial rapport was quickly established. In first interview, he volunteered information on some of his sexual aberrations, provided they were to be considered "of the past only," but denied his prison history during three sessions. On being questioned, he sometimes showed surprise but never hostility. He spoke confidentially and with dramatic inflection, stammering at first, but later only rarely. Characteristically, he clutched the bottoms of his pajamas as though afraid they would drop, not releasing his hold even while lying in bed in the electroencephalographic laboratory. The lack of cooperation referred to in the electroencephalographic report was entirely involuntary; he enjoyed nearly all the tests and offered no resistance to the cystoscopic examination or to the pneumoencephalographic study, from which he suffered only a mild headache of a few hours' duration. He smoked almost incessantly but not more than a third of a cigaret at a time. He wrote brief letters daily to the same few people. In nearly all letters he followed the statement that he felt fine with an urgent appeal for 15, 25 or 50 cents or, in the case of a relative, for $1.00.

More often than not, he was mildly euphoric, exhibiting joy when offered an orange or a piece of candy. Next to eating, he was happiest singing and playing operatic arias, old waltzes and hymns on the piano before an audience. In general, he performed creditably, even though he never remembered the words and sometimes fumbled at the keyboard, lapses which he concealed by humming and improvising finales. His euphoria, however, occasionally gave way to anxiety. When electroshock was being given in the next room, he sometimes walked about, murmuring, "Oh, this is terrible; this is terrible!" On other occasions, he would complain bitterly, "There is nothing to do. I get so bored." Annoyed by patients who teased him about his appetite, he sulked. Such phases were quickly dispelled by giving him attention. He slept well and had no somatic complaints, although his trunk contained sodium bicarbonate, acetylsalicylic acid and rectal suppositories. While his affect was notably shallow, psychomotor activity was neither notably retarded nor accelerated. When called, he motioned vigorously for the doctor or nurse to come to him, obviously hating to move, but showed no laziness in occupational therapy activities, in which he did well. The prospect of almost any new experience thrilled him. Alert, he had an eager interest in his surroundings.

His thought centered on food even after he had regained his weight. Habitually, he would interrupt conversations to ask the time because, as he said, "I want to

know how near dinner it is." The diary he kept on the ward mentioned food at least twice daily, while the nurses' notes referred to his bulimia throughout his stay in the hospital. Returning from supper, he was noted as saying, "Oh, well, it's only fourteen hours to breakfast." Preoccupations other than those with food were not directly reflected in his diary. When told to write out his thoughts, sexual and asexual, as well as the events of the day, he wrote, "No sexual thoughts" repeatedly. For variety, he added: "No sexual trouble," "No trouble sexually," "no unusual thoughts" or "no unnecessary sexual thoughts." For an unknown length of time, his mother and Christ had been telling him "to be of good cheer"; sometimes they ordered him to stop masturbating. Often he saw as well as heard them. Once he showed a flash of insight: "But they're dead. Christ is dead, and mother is dead." Perplexed, he finally broke into a smile, saying, "But their spirit lives on."

He was superficially oriented but believed he had been in the ward six days, when it was actually two; when moved to another closed ward a week later, he was bewildered for several days, not understanding why things looked different. Notwithstanding his conversion to Catholicism prior to 1939, his 1943 diary indicated only attendance at the Baptist Church. On the ward he attended mass regularly, but for a time forgot his conversion: "No, I'm not a Catholic. How silly of me. I am a Baptist, but I go to the Methodist Church. But I have a friend who is a Catholic." Other evidence of loss of memory for recent events, sometimes with compensatory confabulation and suggestibility, was abundant. The morning following the initial two hour interview, he suddenly remarked, "Haven't we met somewhere before?" His attempts to give his work history since his release from prison were completely confused. Jobs, places and people in New York were confounded with those in Chicago. Because he had no idea where it was, extensive search was needed to locate his trunk. Memory for remote events, however, was relatively well preserved, as indicated by the remarkable similarity in the preoperative histories obtained more than five years apart by us. While he showed mild circumstantiality, perseveration and fluctuation of attention were pronounced. At a staff meeting, while talking of his hallucinations, he announced during a pause, "I don't like white milk." When asked what prompted this remark, he replied, "I love chocolate milk."

His vocabulary indicated a better than average intelligence which had deteriorated. His judgment was childish; he was concerned only with his day to day existence. Insight, likewise, was absent. While willing to grant that dates sometimes confused him, he considered his mind sound and his hallucinations "a gift of God." Whenever he was asked about the lobotomy, he typically replied, "It was a perfect success—no desire for the homosexual."

Physical Examination.—After his gain in weight, J. S., at 59, appeared younger than in a photograph taken before operation, seven years earlier. On admission he weighed 115 pounds (53.5 Kg.), but three months later his weight was 156 pounds (70.8 Kg.). Multiple purpuric lesions, ranging from the size of a pinpoint to that of a quarter, with central punctures, suggested bedbug bites. On several examinations fecal matter grossly contaminated his buttocks. Palpation of the skull revealed the trephine holes. The pupils and fundi were normal. His blood pressure was 110 systolic and 76 diastolic, and the pulse rate was 74 per minute. The radial arteries were soft, while the temporal vessels were not prominent or tortuous. The chest, abdomen and genitalia were normal. On rectal examination, the prostate gland felt normal in size and consistency. Both legs and feet showed moderate edema, coldness, sweating and glossiness of the skin. In addition to these persistent trophic changes, there was a conspicuous

dermatographia. Neurologic examination, furthermore, revealed increased but bilaterally equal deep reflexes, a Hoffmann sign bilaterally and equivocal Chaddock and Babinski signs on the left side. Urinary incontinence was noted on eleven occasions, and fecal incontinence, on four.

Laboratory Data.—On his admission to the veterans' hospital, the red blood cell count was 3,640,000; the hemoglobin content, 11 Gm. per hundred cubic centimeters, and the white cell count, 10,200, with 75 per cent polymorphonuclear leukocytes. Within a month the red cell count was 4,480,000; the hemoglobin content, 14 Gm. per hundred cubic centimeters, and the white cell count, 8,800, with a normal differential count. The serologic reactions were negative, and the spinal fluid was normal. Because the preoperative studies included determinations of the visual fields and the basal metabolic rate, a roentgenographic examination of the chest and an electrocardiographic recording, these studies were repeated. All the results were normal with the possible exception of the values for the basal metabolic rate: + 40, + 35, + 22, + 35 and + 13 per cent. Glucose tolerance tests, using the three hour method and the Exton-Rose technic, gave normal values. Except for the trephine holes, the plain roentgenograms of the skull showed nothing pathologic.

In view of the tentative diagnosis of cerebral arteriosclerosis at the time of admission, ophthalmologic consultation was requested and our impression of a normal fundus was confirmed. The enuresis and edema of the lower extremities made it advisable to consult the urologist, although the results of several urinalyses, as well as values for phenolsulfonphthalein excretion (58 per cent), nonprotein nitrogen (31.4 mg. per hundred cubic centimeters) and blood urea nitrogen (17.2 mg. per hundred cubic centimeters) were essentially normal. No albumin was found in any of the four specimens of urine, and the specific gravity varied from 1.010 to 1.022. Cystoscopic studies revealed enlargement of the lateral lobes of the prostate gland. Although slightly trabeculated, the bladder, as studied in the cystometrogram, was not enlarged. The residual urine measured 2 cc. An intravenous pyelogram showed normal kidneys, calices, pelves and ureters, with the albumin: globulin ratio (1.75) were determined when the patient weighed 138 pounds (62.6 Kg.). While these values for protein were normal, the bipedal edema was just as pronounced as on the day of the patient's admission to the psychiatric ward, a month earlier, when he weighed 120 pounds (54.4 Kg.).

Electroencephalographic Data.—Dr. F. A. Gibbs and Mrs. E. L. Gibbs made the following report:

"Several routine electroencephalograms showed low voltage, fast activity with a great deal of low voltage, irregular slowing in both frontal areas, possibly more prominent on the left.

"Waking Electroencephalogram: The record was suggestive of a bifrontal focus of slow waves, more pronounced on the left. Because the patient was uncooperative, it was thought advisable to obtain a sleeping record in order to rule out movement artefacts.

"Sleeping Electroencephalogram: Two electroencephalograms, taken during anesthesia induced with 'pentothal' N.N.R., showed scattered spike seizure discharges in the left frontal area and extreme asynchronism between the two frontal regions. Other leads showed normal sleep activity. The left frontal lead showed large, irregular, slow waves, while few such waves were evident in the right frontal area. However, there was less normal 14 per cent activity in the right frontal area. In the records of 98 control subjects no asynchronism was observed in the frontal areas during sleep.

TABLE 1.—*Summary of Psychometric Data*

Name of Test	Results of Tests
Army Alpha (Form A) Bregman	MA equivalent = 16 yr. 5 mo. (score = 92) IQ equivalent = 109 (15 yr. basis)
Wechsler-Bellevue Intelligence Scale	Verbal Tests: Information 12; Comprehension 10; Digits 10; Arithmetic 7; Similarities 8; Vocabulary 13 Performance Tests: Performance arrangement 6; Performance completion 8; Block design 3; Object assembly 10; Digit symbol 9 Verbal IQ = 106 Performance IQ = 105 Full scale IQ = 105 Impairment = 28% Impairment for age = 16% Net impairment = 12% Wechsler states that more than 10% impairment indicates "possible deterioration"
Wechsler Memory Scale	Memory quotient = 94 Normal MQ = full scale IQ = 105 Memory loss = 10% beyond that expected for age
Minnesota Vocational Test for Clerical Workers	Number comparison = 10th percentile Name comparison = 27th percentile (using norms of employed clerical workers)
Bender Gestalt Test	"Showed little that was significant"
Words named in three minutes (Worchel, P., and Lyerly, J. G.: J. Neurophysiology 4 : 62 [Jan.] 1941)	81 words (60 words is passing in this test at the 10 year level of the 1916 Stanford-Binet test. Failure in this test has been reported for other lobotomized patients)
Goldstein-Scheerer Cube Test	"In designs I to IX and XI he succeeded on the first trial, but in designs X and XII he succeeded only on the second trial, was slow and showed difficulty in breaking the design into its parts, pointing to possible organicity."
Weigl-Goldstein-Scheerer Color Form Sorting Test	"He sorted quickly and without error but showed tendency toward concrete behavior characteristic of damage to the brain in that he arranged the figures in a pattern."
Haufmann-Kasanin (Vigotsky Blocks) Test	"The patient showed the lack of associative thinking and ability to synthesize and inability to carry out ideational drives characteristic of damage to the brain."
Babcock-Levy Examination of Efficiency of Mental Functioning (short form, as used by Rapaport)*	Learning.....Average score = 9 Repetition.....Average score = 14 Motor.....Average score = 14 Total average score = 12.3 Vocabulary: MA = 17.6 Efficiency index = —5.3 An index below —3 indicates profound impairment, but the test makes no adjustment for age
Robinson "Deliberation Tests"	Rhymes = 6 Numbers = 3 Total.... = 9 Average score for control group = 15.43 Average score for lobotomized group = 6.5
Porteus Maze Test (Porteus and Kepner [4])	March 4 MA = 12 yr. March 17 MA = 9 yr. March 26 MA = 12 yr. Nearly all members of control group showed practice effect
Kohs Block Design	Score: 38 MA equivalent: 9 yr. 7 mo.
Knox Cube Test	Score: 6 MA equivalent: 7 yr.

* Rapaport, D.: Manual of Diagnostic Psychological Testing: Diagnostic Testing of Intelligence and Concept Formation, New York, Josiah Macy Jr. Foundation, 1944, p. 143.

"Impression: The electroencephalogram was strongly suggestive of disorder in both frontal areas, with epileptic diasthesis."

Psychometric Data.—Thanks to the generous cooperation of the Department of Psychology, Veterans Administration Facility, Hines, Ill., headed by Dr. Roy Brener, J. S. was given a battery of tests. In addition to the Army Alpha Test, for which a preoperative score was available, the Robinson [3] and Porteus [4] tests were selected because their originators have found them useful in differentiating lobotomized patients from a control series. A clerical test was given to see whether the patient's work deterioration was based on a decline of specific work ability. Ten additional tests were chosen because of their reported sensitivity in

TABLE 2.—*Summary of Comparable Preoperative and Postoperative Data*

	Status Up to Nov. 1941	Status After Nov. 1941
Symptoms	1. Insomnia 2. Constipation 3. Headaches, many and severe 4. Mild stammer	1. No insomnia 2. Less constipation 3. Headaches, few and mild 4. Mild stammer 5. Bulimia 6. Occasional incontinence
Work history	Kept no job less than 1 year Kept one job 18 years	1943: Kept no job more than 9 mo. 1944: Kept no job more than 3 mo. 1945: Odd jobs only 1946: No jobs
Psychosexual behavior	Overtly masochistic, pedophilic and homosexual; daily masturbation	Preoccupied with same sexual fantasies, but probably with decreasing overt activity
Mental status	October 1940 to November 1941 1. Agitation, tension, apprehension 2. Depression 3. Obsession and compulsion (to masturbate) 4. Conscientiousness (in work) 5. Timidity and anxiety 6. Sensorium intact 7. No hallucinations 8. Insight and judgment fairly good	January 1947 to April 1947 1. Confusion 2. Mild euphoria and shallow affect 3. Irresponsibility 4. Childishness 5. Desire for attention 6. Memory loss, confabulation, suggestibility, circumstantiality, perseveration and fluctuation of attention 7. Hallucinations 8. No insight, poor judgment
Physical examination	Weight: 134 pounds Neurologic status normal	Weight: 115 → 156 pounds Increased deep reflexes Bilateral Hoffmann sign Equivocal Babinski and Chaddock signs on left Trophic changes both lower extremities
Army Alpha test	August 1939 Mental age: 17-11 Intelligence quotient: 119	March 1947 Mental age: 16-6 Intelligence quotient: 109

cases of damage to the brain. The results are given in table 1, arranged so that the first eight are the tests showing minimal impairment and the remaining six the tests which may be considered significant.

E. SUMMARY OF COMPARABLE PREOPERATIVE AND POSTOPERATIVE DATA

In table 2 we have collected all the information concerning the patient in which direct comparison can be made between his preoperative and

3. Robinson, M. F.: What Price Lobotomy? J. Abnorm. & Social Psychol. **41**:421 (Oct.) 1946.

4. Porteus, S. D.: Medical Applications of the Maze Test, M. J. Australia **1**:558 (June 17) 1944. Porteus, S. D., and Kepner, R. D.: Mental Changes After Bilateral Prefrontal Lobotomy, Genet. Psychol. Monogr. **29**:4 (Feb.) 1944.

his postoperative status. Certain data, such as the physical and laboratory findings that showed no significant differences between 1941 and 1947 are not mentioned. Excluded, too, are the basal metabolic rates, since the five readings in February 1947 were too variable to be considered reliable. Probably the lowest figure, +13 per cent, alone is valid, in which case the preoperative reading, +14.5 per cent, indicates that no change had occurred.

COMMENT

While it is not our major problem, we shall formulate briefly the psychopathologic features of our case prior to the lobotomy. With a remote, weak, unloved husband, a dominant, demonstrative wife was led to seduce their son. She periodically slept with the boy until he was 10 years of age and kissed him after each spanking. Her spanking-kissing acts eroticized his buttocks, and her applications of oil for pinworms (and the pinworms themselves) eroticized his anus. As though her spankings (eventually terminating regularly with ejaculation) were not incentive enough for the patient to act naughty, his father rewarded the spankings with money. His mother's dominance explains the patient's feminine identification; the intense rewards for misbehavior, his psychopathic personality. In the puritanic environment, however, his weak superego ultimately grew strong enough to rebel against further spanking intimacies with his mother. Thereafter, his homosexual gratification was sought in a masochistic and pedophilic form because he remained emotionally fixed at the age level at which his mother provided gratification. Imprisoned at the age of 52 for a pedophilic act, he became the victim of an obsession that on release he would commit a child rape punishable by death. The sadism invariably associated with masochism may have been reenforced by involutional changes and perhaps directed against the child in revenge for his incarceration. At any rate, he presented, overlying his psychopathic personality, a neurosis engendered by the conflict between these new rape-murder impulses and his superego. This led to the symptoms of panic and obsession. The lobotomy was seized on by him as an appeasement to his superego, and conceivably as the ultimate of spankings.

Our primary concern is with the effects of the lobotomy on the patient. It is imperative that we determine whether the postoperative changes in the patient were actually due to the lobotomy. There is suggestive evidence that cerebral arteriosclerosis may have been at least a complicating factor. The patient's father died of asthma, presumably cardiac; the mother, of cerebral hemorrhage, and the grandfather, of a psychosis with cerebral arteriosclerosis. The organic psychosyndrome and the pneumoencephalograms are compatible with such a diagnosis. These facts, however, lose their significance when we recall that the cardiac symptoms of the father began at the age of 75, the mother's

stroke at 82 and the psychosis of the grandfather at 93. The organic syndrome, and the underlying atrophy of the brain, moreover, are common to several diseases of the brain and can hardly be used as evidence for the symptoms belonging to any one of them. On the other hand, there are specific findings that are not ordinarily compatible with a diagnosis of cerebral arteriosclerosis. The retinal vessels, the peripheral vessels, the heart sounds, the roentgenographic configuration of the heart, the electrocardiogram and the blood pressure were all normal, whereas the explosive emotional reactions, restlessness, insomnia at night, hypersomnia in the daytime, gait, somatic complaints and focal symptoms common to that disease were all absent. On the basis of the available evidence, then, a diagnosis of cerebral arteriosclerosis is not tenable.

The pneumoencephalograms, except for the trephine holes, could just as well be that of a patient with presenile psychosis. If either Alzheimer's disease (presenile sclerosis) or Pick's disease complicated the picture, however, one would expect aphasia, agnosia, apraxia and either psychomotor acceleration and agitation (Alzheimer's disease) or psychomotor retardation and apathy (Pick's disease), none of which was present in our patient. Stengel [5] speculated that lobotomy in a typical case of Alzheimer's disease would lead to the development of Pick's syndrome. In accordance with this speculation, there is the possibility that the agitation of our patient the year prior to lobotomy was based on the presence of Alzheimer's disease, which the lobotomy transformed into Pick's syndrome. A similarity exists between the effects of lobotomy and Pick's disease which is hardly surprising, since both attack the white matter of the frontal lobe. But to postulate the existence of a rare disease, and thereby leave unexplained the negative findings, in preference to using the lobotomy, which alone can explain all the findings, is to violate the principles of logic. Finally, we must mention the diagnosis of involutional melancholia as not entirely inconsistent with the picture that the patient presented prior to lobotomy. Ordinarily this disease is not considered a degenerative disease of the brain; in fact, Freeman and Watts have consistently reported some of their best results in cases of this type. If, on the other hand, some cases of involutional depression are really cases of Alzheimer's disease, as Stengel asserted, then our statements on the presenile psychoses still apply. Again, if we assume that the involutional changes were manifestations of cerebral arteriosclerosis, we need not repeat what we said concerning that disease. Theoretically, involutional melancholia may, in some cases, be the syndrome of a degenerative disease of the brain not yet defined. To explain our observation in this way, however, is to ignore the law of parsimony.

5. Stengel, E.: A Study of the Symptomatology and Differential Diagnosis of Alzheimer's Disease and Pick's Disease, J. Ment. Sc. **89**:1 (Jan.) 1943.

To rule out any complicating disease, it is necessary to explain all the findings in our patient in terms of the lobotomy. Freeman [6] has conveniently given a table which lists, first, the symptoms relieved and, second, the symptoms produced by lobotomy. In the first list are noted apprehension, anxiety, depression, insomnia, agitation, obsessions, panic and compulsions. These symptoms also describe the mental status of our patient during the year preceding lobotomy and were permanently absent thereafter (table 2). On the other hand, the table listing symptoms produced by lobotomy includes the following symptoms, which our patient showed only after lobotomy: emotional flattening, poor judgment, euphoria, perseveration, confabulation, gain in weight, increased appetite, neurologic changes and incontinence. With more cases, Watts and Freeman [7] published another list of persistent symptoms produced by the operation. Those which describe our patient further are lack of initiative, circumstantiality, suggestibility and fluctuation of attention. Later, these authors reported that psychopathic and homosexual persons are not benefited,[8] having already indicated that sexual activity is little altered by lobotomy.[9] Again this observation is in keeping with the status of our patient, who roamed the parks and wrote pornographic notes after operation, although no doubt his overt sexual activities declined consistent with his age. Even the 18 month drop in mental age in the Army Alpha Test, half of which, according to Wechsler,[10] is normal for his age, is consistent with their statement, "Formal intelligence tests reveal little or no defect."[11] Finally, they referred repeatedly to the childishness in their patients.[12] Childishness was prominent in our patient and helps to explain his constant desire for

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6. Freeman, W.: The Surgical Treatment of Mental Disorders, M. Ann. District of Columbia **8**:345 (Dec.) 1939.

7. Watts, J. W., and Freeman, W.: Intelligence Following Prefrontal Lobotomy in Obsessive Tension States, J. Neurosurg. **1**:291 (July) 1944.

8. Watts, J. W., and Freeman, W.: Prefrontal Lobotomy: Factors Influencing Prognosis, South. Med. & Surg. **108**:241 (Aug.) 1946.

9. Freeman, W., and Watts, J. W.: Prefrontal Lobotomy: Convalescent Care and Aids to Rehabilitation, Am. J. Psychiat. **99**:798 (May) 1943.

10. Wechsler, D.: Measurement of Adult Intelligence, ed. 3, Baltimore, Williams & Wilkins Company, 1944, p. 226.

11. Freeman, W., and Watts, J. W.: The Radical Treatment of the Psychoses and Neuroses, Dis. Nerv. System **3**:6 (Jan.) 1942.

12. Freeman, W., and Watts, J. W.: (*a*) Psychosurgery: Intelligence, Emotion and Social Behavior Following Prefrontal Lobotomy for Mental Disorders, Springfield, Ill., Charles C Thomas, Publisher, 1942; (*b*) footnote 11; (*c*) Prefrontal Lobotomy: The Surgical Relief of Mental Pain, Bull. New York Acad. Med. **18**:794 (Dec.) 1942; (*d*) footnote 9; (*e*) Prefrontal Lobotomy: An Evaluation of Two Hundred Cases over Seven Years, J. Ment. Sc. **90**:532 (April) 1944; (*f*) Prefrontal Lobotomy: Survey of Three Hundred Thirty-One Cases, Am. J. M. Sc. **211**:1 (Jan.) 1946; (*g*) Prefrontal Lobotomy: Indications and Contraindications, read before the Chicago Neurological Society, Feb. 11, 1947.

attention and his irresponsibility. It is their finding that with time and training this childishness diminishes and the work history improves over the preoperative level. But they reported:

Some patients have given every evidence of complete and permanent disability up to the present time several years after placement of incisions too far posterior.[12f]

Since the trephine holes were placed by an eminent neurosurgeon at the site that Freeman and Watts themselves recommended (figure *B*), we ascribe the childishness and the deterioration in the work history of our patient to the operation rather than to the surgeon.

For the remaining symptoms, we must go to other authors. The loss of memory which was so prominent in our case was stressed only by Hutton:[13]

. . . . after the operation reproductive memory or recall is to a large extent replaced by associative memory, and the patients live in a perpetual present unable to take thought for tomorrow.

Cohen[14] and Hutton, Fleming and Fox[15] noted persistent foci of asynchronous, slow waves in the frontal area, similar to those in our patient's electroencephalogram. The trophic changes in the lower extremities were reported by Ziegler and Osgood[16] in 8 of 19 patients. It is probable that the cellulitis of the left foot and ankle, for which our patient was originally admitted to the hospital, was on this basis. This is suggested by the fact that the infection occurred on the left side, on which he exhibited the Chaddock sign, whereas the atrophy of the frontal cortex and the enlargement of the anterior horn were greater on the right. The latter changes, in turn, may have been related to the operative procedure in which the right horn alone was entered. Why the electroencephalogram suggests greater damage on the left we do not know.

We have explained all the findings, except the hallucinations and the cerebral atrophy, in terms of observations on other lobotomized patients. While the few available reports indicate localized atrophy of the dorsomedial nucleus of the thalamus (Freeman and Watts[12e]) and of the frontal cortex (Meyer and Beck[17]), no case comparable to ours has appeared. This is not surprising, since autopsies or air studies made at long intervals after the operation have been too few to make valid any generalization concerning the long term effects of lobotomy on the

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13. Hutton, E. L.: The Investigation of Personality in Patients Treated by Prefrontal Leucotomy, J. Ment. Sc. **88**:275 (April) 1942.

14. Cohen, R.: Electroencephalographic Study of Prefrontal Lobotomy, Arch. Neurol. & Psychiat. **53**:283 (April) 1945.

15. Hutton, E. L.; Fleming, G. W., and Fox, F. E.: Early Results of Prefrontal Leucotomy, Lancet **2**:3 (July 5) 1941.

16. Ziegler, L. H., and Osgood, C. W.: Edema and Trophic Disturbances of the Lower Extremities Complicating Prefrontal Lobotomy, Arch. Neurol. & Psychiat. **53**:262 (April) 1945.

17. Meyer, A., and Beck, E.: Neuropathological Problems Arising from Prefrontal Leucotomy, J. Ment. Sc. **91**:411 (Oct.) 1945.

brain. The frontal lobes connect not only with the dorsomedial nucleus of the thalamus but with all the rest of the brain. Theoretically, other parts may degenerate after local trauma. Severe generalized atrophy sometimes follows head trauma when the brain is injured, and lobotomy is, after all, an injury to the brain with sequelae common to it. Thus, Watts and Freeman [8] reported convulsive seizures in 10 per cent of their cases. Our patient's electroencephalogram, indicating an "epileptic diathesis," is indistinguishable from the records in certain cases of head injury with post-traumatic epilepsy. If post-traumatic epilepsy has its counterpart in postlobotomy epilepsy, perhaps post-traumatic psychosis also has a counterpart in which hallucinations may play a role. Their statement,[12a] "Without the frontal lobes there could be no functional psychoses," is not applicable, since our patient has an organic, and not a functional, psychosis.

Freeman and Watts,[11] in referring to the pre-Moniz studies of damage to the frontal lobe—the famous Crowbar case, tumor, and cases of war injuries and lobectomy—stated:

> Out of these studies has come the picture of the "defrontalized dement," a being without shame and without ambition, lacking in all the finer qualities that make a man what he is, and debased to the level of the brute.

While they added that such a picture does not follow lobotomy, they indicated the theoretic basis for such a possibility. We would stress that these early studies show the effects of damage to the frontal lobe in nonpsychotic patients, whereas the work of Freeman and Watts has been on mentally ill patients, in whom the development of a new psychosis might go unrecognized or be noted merely as a condition preferable to the original psychosis. But even in the literature on effects of the operation on the mentally ill patient, there have been hints that the "defrontalized dement" is more than a name. Thus, Ström-Olsen and associates [18] described the case of a lobotomized patient who "displayed the features of an organic dementia characterized by amnesia, confusion, marked euphoria with emotional facility and childishness." Already, several other investigators have reported cases in their series in which the condition was made worse by the operation (Govindaswamy and Rao,[19] Heilbrunn and Hletko,[20] Hletko and associates,[21] and McKis-

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18. Ström-Olsen, R.; Last, S. L.; Brody, M. B., and Knight, G. C.: Results of Prefrontal Leucotomy in Thirty Cases of Mental Disorder, with Observations on Surgical Technique, J. Ment. Sc. **89**:165 (April) 1943.

19. Govindaswamy, M. V., and Rao, B. N. P.: Bilateral Prefrontal Leucotomy in Indian Patients, Lancet **1**:466 (April 8) 1944.

20. Heilbrunn, G., and Hletko, P.: Disappointing Results with Bilateral Prefrontal Lobotomy in Chronic Schizophrenia, Am. J. Psychiat. **99**:569 (Jan.) 1943.

21. Hletko, P.; Baer, W., and Schlan, L.: Review of the Status of a Series of Lobotomies After a Lapse of Five Years, with Motion Pictures, read before the Illinois Psychiatric Society, Feb. 6, 1947.

sock [22]). The contention that such cases demonstrate faulty technic is beside the point, since even with the Lyerly method the operation remains fundamentally blind.

Considering the obviously deteriorated status of the patient by clinical standards, the dearth of positive findings in psychologic tests selected solely to bring it out is amazing. In table 1, the first eight tests show little or no impairment beyond what is to be expected for a person of the patient's age, but the results of the last six tests are probably significant. Because we have no preoperative data, we can merely state that an adult with a mental age on the Army Alpha Test of 16 years 5 months (and a Wechsler intelligence quotient of 105) probably shows impairment, if in the Kohs Block Design Test he functions at a 9 year 7 month level and in the Knox Cube Test, at a 7 year level. The results with the Porteus Maze Test were positive in that he, in common with other lobotomized patients, acted like the feebleminded group in showing no practice effect. Robinson's Deliberation Tests, designed to differentiate lobotomized and nonlobotomized patients, placed our patient with the lobotomized group. The Vigotsky Blocks Test and the Babcock-Levy Test showed significant changes. Since the results of these tests, purporting to measure ability in abstraction (Vigotsky Blocks and Kohs Block Design Test), immediate memory (Knox Cube Test), sustained attention (Robinson Deliberation Tests), ability to gain from experience (Porteus Maze Test) and intellectual deterioration (Babcock-Levy Test), fit the clinical picture, they are regarded as significant.

The relative paucity of psychometric abnormalities in view of the clinical picture is consistent with the reports in the literature. With the exception of Porteus and Robinson, no other investigators using a control group have been able to demonstrate the effects of lobotomy under standard test conditions. This anomalous situation deserves attention. Cobb [23] suggested that the functional disturbance before lobotomy is replaced by an organic defect afterward. If, in combining the results in a large series of cases, the postoperative deficit is found to equal the preoperative impairment, the conclusion must be, "There is no evidence of appreciable change in general mental ability" (Freeman and Watts [12a]). Until tests are made which clearly distinguish functional and organic impairment, this conclusion is inevitable. The lack of significant test results may well be due to a lack of significant tests. Since two psychologists have shown that the proper tests yield meaningful results, perhaps others will invent or select appropriate test material.

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22. McKissock, W.: The Technique of Prefrontal Leucotomy, J. Ment. Sc. **89**:194 (April) 1943.

23. Cobb, S.: Review of Neuropsychiatry for 1940, Arch. Int. Med. **66**:1341 (Dec.) 1940.

Recently Freeman and Watts [12g] presented the indications and contraindications to lobotomy before the Chicago Neurological Society. Freeman stated the belief that psychopathic personality is a contraindication to the operation, but specified as ideal a patient with an obsessive-compulsive neurosis who is past middle life and had a good work record and educational history. On this basis, one could predict what actually happened in our case—the lobotomy relieved the patient of his obsessive state without improving, fundamentally or permanently, his psychopathic personality. On the other hand, Freeman added that their "surgically induced children" continue to grow up and improve for five or more years after operation. Instead of progressive improvement, our patient showed rapid improvement after the immediate postoperative period, stabilization for a year and then progressive decline. Lobotomized in November 1941, he was first recognized as psychotic in March 1945 and demented in January 1947. Since there is no evidence for any complicating factor, and we can explain all our findings in terms of the effects of the operation itself, we conclude that the lobotomy produced the dementia.

NOTE: Dr. Samuel J. Beck administered the Rorschach test to the patient, but his results were not available until after this paper went to press. A Rorschach test made a few months after operation by Dr. Harrower-Erickson gave no indication of cerebral damage, showing instead a fairly healthy picture (Banay and Davidoff [2]). Beck's findings, five years later, were consistent with results of other psychometric devices in showing that any abnormalities were relatively mild as compared with the clinical picture. It is pertinent, nevertheless, to give his summary. "The reaction pattern appears characteristic for patients with a pathologic process of the brain which has not gone far, since a residue of higher functioning persists."

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TENSILE STRENGTH OF HUMAN NERVES

An Experimental Physical and Histologic Study

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GRAFTS of peripheral nerves across long gaps, except for the facial and the short finger nerves, were a dismal failure in the recent war, as they had been before. This was true of fresh homologous grafts (Spurling and associates [1]) and of our own attempts with dehydrated frozen grafts (Nulsen, Lewey and Van Wagenen). This sad experience, together with the urgent need of some kind of nerve repair, focused attention on the possibility of end to end sutures after extensive mobilization and transposition of the corresponding nerves in conjunction with maximal flexion of the extremities. Encouraging results with this procedure were reported by Naffziger,[3] Platt,[4] Platt and Bristow,[5] Babcock,[6] Forrester-Brown [7] and, lately, Highet and Holmes.[8] The

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From the Neurological and Neurosurgical Sections of Cushing General Hospital, Framingham, Mass., and the Neuropathological Section of the Wallace Research Laboratory, Wrentham, Mass.

1. Spurling, R. G.; Lyons, W. R.; Whitcomb, B. B., and Woodhall, B.: Failure of Whole Fresh Homogenous Nerve Grafts in Man, J. Neurosurg. **2**:79 (March) 1943.

2. Nulsen, F. E.; Lewey, F. H., and Van Wagenen, W. P.: Experiences with Peripheral Nerve Grafting, Surgeon General's Medical History of World War II: Neurosurgery, to be published.

3. Naffziger, H. C.: Methods to Secure End-to-End Suture of Peripheral Nerves, Surg., Gynec. & Obst. **32**:193 (March) 1943.

4. Platt, H.: Surgery of Peripheral Nerve Injuries of Warfare, Brit. M. J. **1**:596 (April 23) 1921.

5. Platt, H., and Bristow, W. R.: The Remote Results of Operations for Injuries of Peripheral Nerves, Brit. J. Surg. **11**:535 (Jan.) 1924.

6. Babcock, W. W.: Standard Technique for Operations on Peripheral Nerves, with Special Reference to Closure of Large Gaps, Surg., Gynec. & Obst. **45**: 364 (Sept.) 1927.

last two authors followed the course of 42 patients operated on during this war at the Oxford Nerve Center for gaps of 1.5 to 11 cm. in practically all nerves of the upper and lower extremities. More than 90 per cent of these patients have shown some degree of recovery in due time.

Highet and Sanders [9] also examined the question of the upper limit of gaps which can be directly bridged in the hope of good functional results. They came to the conclusion that 9 cm. was the maximum distance over which a human median nerve could be sutured and again stretched without serious damage to both its proximal and its distal stump. Similarly, the maximum gap for a peroneal nerve was 11 cm. They made, furthermore, a systematic investigation of the injuries of the lateral popliteal nerve of dogs caused by traction after suture. They found that elongation of nerves up to 6 per cent of their total mobilized length did not damage either the central or the distal stump. Elongation of 11 per cent or more caused extensive degeneration in the central stump, rupture of Schwann tubules, damage to blood vessels and the appearance of patches of fibroblasts surrounded by collagen in the peripheral stump.

Denny-Brown and Doherty [10] experimentally elongated the peroneal nerve of living cats about 100 per cent of its original length. The nerve extended "moderately easily, in the manner of a plastic material, . . . often without any signs of injury. . . . Recovery of apparently full power of dorsiflexion of the foot and spreading of the toes occurred within fourteen days." With such extension, some of the larger axis-cylinders became beaded. If the nerve was extended beyond 100 per cent of its resting length, smaller epineural vessels were torn and the nerve fibers damaged by ischemia, manifested by patchy edema and loss of segments of the myelin sheath. "The neural degeneration thus produced is recoverable without complication." The maximum degree of disorder of nerve in continuity was seen in connection with the appearance of a longitudinal split in the perineurial sheath with herniation of the nerve bundles. In conclusion, minor histologic damage and full functional recovery followed experimental extension of the peroneal nerve by 100 per cent of its resting length in the living cat.

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7. Forrester-Brown, M. F.: The Possibilities of Suture After Extensive Nerve Injury, J. Orthop. Surg. **3**:277 (June) 1921.

8. Highet, W. B., and Holmes, W.: Traction Injuries to Lateral Popliteal Nerves and Traction Injuries to Peripheral Nerves After Suture, Brit. J. Surg. **30**:212 (Jan.) 1943.

9. Highet, W. B., and Sanders, F. K.: Effects of Stretching Nerves After Suture, Brit. J. Surg. **30**:355 (April) 1943.

10. Denny-Brown, D., and Doherty, M. M.: Effects of Transient Stretching of Peripheral Nerve, Arch. Neurol. & Psychiat. **54**:116 (Aug.) 1945.

This observation brings up the question of the tensile strength of human nerves. Tillaux [11] stretched the median and ulnar nerves of human cadavers and reported that they could stand an extension of 15 to 20 cm. (about 34 to 45 per cent of their length) and were not grossly ruptured until a weight of 20 to 25 Kg. had been applied. This and similar old statements were of little help in our problem, since they measured the force actually rupturing the entire nerve, and not the beginning of damage to single nerve fibers. We felt, therefore, that we should measure the tensile strength of human nerves with modern methods, determine their stress-strain curves and examine the degree of their histologic damage appearing at the critical points of the curves.

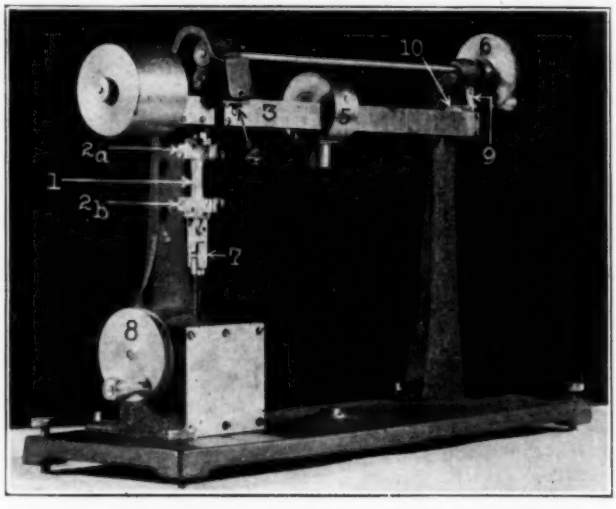

Fig. 1.—The tensiometer. See text for explanation.

METHODS AND MATERIAL

Technic.—A commercial tensiometer was used (fig. 1).[12] This apparatus takes about a 4 cm. length of tissue (*1*) between two clamps (*2a* and *2b*). The upper clamp (*2a*) hangs from the short end of a metal beam (*3*), which, in turn, is suspended from a fulcrum (*4*). The beam is balanced by means of a weight (*5*), which moves on the long arm of the beam with the help of a wheel (*6*). The lower clamp (*2b*) is set on top of a jack (*7*), which is moved up and down by turning the handle of another wheel (*8*). To prevent slipping, the nerve ends are wrapped in a fine layer of cotton and gauze and fixed in the clamps (*2a* and *2b*) while the weight (*5*) is at zero. The nerve is stretched by moving down the clamp (*2b*) by means of the wheel (*8*) until the tongues (*9*) are balanced. This is considered the

11. Tillaux, P.: Des affections chirurgicales des nerfs, Paris, P. Asselin, 1866, p. 11.

12. The Harrison Department for General Surgical Research, of the University of Pennsylvania School of Medicine, permitted us to use its apparatus for this study.

resting length of the nerve. Markers are placed on the sheath of the nerve to control the actual elongation of the nerve with the help of a caliper.

The experiment is performed as follows: The nerve is elongated 0.07 mm. by turning the handle (*8*) 360 degrees. The resulting tension is measured by moving the weight (*5*) with the help of a wheel (*6*) until the tongues (*9*), displaced by the elongation of the nerve, are balanced again. The degree of tension is read off the beam in kilograms to the left of the weight and in grams on the micrometer wheel (*10*). When the tongues have remained stable for at least one minute, the lower clamp is moved down another 0.07 mm. This process is repeated until the nerve is stretched to the desired degree. At that time the nerve is removed from the clamps, placed on cardboard and fixed in solution of formaldehyde U.S.P. (1:4) for histologic examination.

Material.—The material consisted of 22 human nerves—3 sciatic, 4 ulnar, 7 tibial and 8 peroneal nerves. The nerves, with few exceptions, were obtained (and used) within six to twelve hours after death from patients who had died of diseases which, in all probability, could not have had any relation to the condition of the nerves.

The histologic picture illustrates the condition of the nerves at the end of the experiment and represents the maximal elongation or tension of the nerves. Although there is definite interest in this stage of the experiment—as will be seen in the histologic description—the practical issue for which the examination was undertaken is concerned with the other end of the range, namely, with the minimal permissible tension above which the first tear of nerve elements may be expected. Developments beyond our control prevented our continuing the experiments to the point where the histologic examination would fail to show pathologic changes.

RESULTS

The problem was first approached from the physical angle. Stress-strain curves were drawn for 7 technically good experiments—4 on the ulnar and 3 on the peroneal nerve (fig. 2). Disregarding the shape of the curves between 0 and 1 per cent of elongation, for which no useful data could be obtained with our apparatus, all curves showed basically the same characteristics: a rise in a straight line, or a slightly parabolic curve, to a point where it suddenly changed its slant to a sharply steeper ascent.

According to Hooke's law, the increase of stress is proportional to that of strain within the limits of the elasticity of a material. Hence, a straight line will result from plotting the values of stress and strain in a coordinate system. The point where the straight line becomes discontinuous indicates that the material has been stretched beyond the limits of its elasticity and has probably become plastic. If the material is not uniform, but consists of tissues with different modulus of elasticity, such as the comparatively soft axis-cylinder and myelin, on the one hand, and the much harder mesenchymatous endoneurium and perineurium, on the other, it is possible that the first becomes plastic, whereas the latter resists longer and finally ruptures.

Stress-strain curves for plant and animal tissues suggest that their elasticity is rubber-like for soft tissues, such as the stem of plants or

the gut and tendons of animals. Wooden roots of plants and bones, on the other hand, show a metal-like elasticity. Stress-strain curves for rubber-like material are *S* shaped; those of metal-like material approximate a straight line (Treitel [13]). The uncertainty of the actual shape

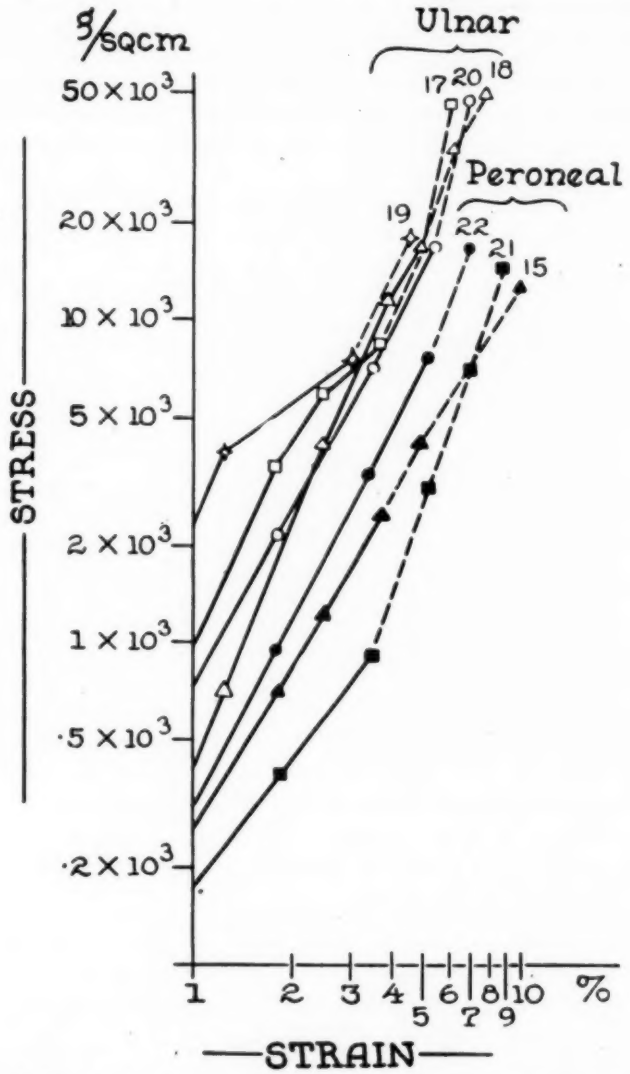

Fig. 2.—Stress-strain curves for 4 ulnar and 3 peroneal human nerves. Strain means the elongation of the nerve, expressed, in percentages of its total length. Stress means the tension of the nerve at the corresponding elongation, expressed in grams per square centimeter of the cross section area of the nerve. No useful values could be obtained in the range below 1 per cent strain. We purposely omitted fitting curves to the determined points below this point because of lack of sufficient data in the neighborhood of the break in the curve. The lines of dashes indicate the portions of the stress-strain curve after they have become discontinuous. This is construed to indicate the area where the first nerve elements rupture.

13. Treitel, O.: Elasticity of Plant Tissues, Tr. Kansas Acad. Sc. **47**:219, 1944.

of the curves below 1 per cent in our own, as in any previously investigated, material prevents any statement on this point, although it is unlikely that nerve tissue should have the elasticity of metal.

Returning, after this digression, to our own curves, it may be stated with certainty that something happens to the nerve tissue at the breaking point of the curves, and it is likely that the discontinuity indicates damage to the nerve.

TABLE 1.—*Details of Stress and Strain Curves for Seven Human Nerves*

Experiment No.	Nerve	Area of Cross Section, Sq. Cm.	Percentage of Elongation of Original Length	Tension, Kg./Sq. Cm.	Critical Point	Critical Elongation
15	Peroneal	0.2	1.75	750		
			2.50	1,250		
			3.75	2,500	+	
			5.00	4,250		
			10.0	13,000		
21	Peroneal	0.13	1.8	385		
			3.5	925	+	4.1%
			5.25	3,100		
			7.0	7,300		
			8.75	15,000		
22	Peroneal	0.13	1.8	925		
			3.5	3,460		
			5.25	7,800	+	
			7.0	16,200		
17	Ulnar	0.07	1.8	3,570		
			2.5	6,000		
			3.75	8,860	+	
			5.0	17,100		
			6.0	30,700		
18	Ulnar	0.07	1.25	700		
			2.5	4,300		
			3.75	11,400		
			5.0	17,100	+	4.2%
			6.25	33,000		
			7.5	49,300		
			8.75	67,000		
19	Ulnar	0.07	1.25	3,859		
			3.1	7,715	+	
			4.7	15,286		
20	Ulnar	0.07	1.8	2,150		
			3.5	7,150		
			5.25	17,100		
			7.0	48,000		
			8.75	75,000		

The critical point is located at a mean elongation of about 4.1 per cent for the peroneal and 4.2 per cent for the ulnar nerve, with an average tension of 3.7 and 12.7 Kg. per square centimeter, respectively (table 1). One may, therefore, say that the risk of overstretching these nerves begins at an elongation above 4 per cent of their total length (critical elongation). Combination of the values obtained in all 7 experiments gives a range of 3.2 to 5.2 per cent as that of minimal nerve damage through overstretching, or a mean of 4.2 per cent.

TABLE 2.—*Record of Stretching Experiments and Summary of Histologic Observations*

Experiment No.	Nerve	Area of Cross Section, Sq. Cm.	Final Elongation, %	Final Tension, Kg./Sq. Cm.	Figure Number	Histologic Description
1	Peroneal	0.13	31	?	..	Longitudinal split in perineurium; herniation of nerve fasciculi (pseudoneuroma); complete transverse rupture of myelinated nerve fibers and endoneurial sheath; microscopic tears of individual myelinated nerve fibers and small fasciculi at various levels throughout stretched segment; myelin sheaths partly broken down; conspicuous vacuolation
2	Peroneal	0.13	10	3.0	..	Complete transverse rupture of myelinated nerve fibers and endoneurial sheaths at one point; preservation of perineural tube; microscopic tears of individual myelinated nerve fibers, including endoneurial sheaths and smaller fasciculi at several levels
3	Peroneal	0.13	16	32.0	..	Similar to that in experiment 1
4	Peroneal	0.13	20	53.0	..	Similar to that in experiment 2
5	Sciatic	0.13	21	?	..	Similar to that in experiment 1
6	Sciatic	0.07	10	?	..	Complete transverse rupture at one level as in experiment 2; microscopic tears of individual myelinated nerve fibers and smaller fasciculi; endoneurium squeezed into space and rupture of blood vessels
7	Sciatic	0.07	11	?	..	Same as that in experiment 6
8	Peroneal	0.09	27	14.7	..	Complete transverse rupture of each of three larger fasciculi with preservation of perineurium and some of larger endoneurial septums; each fasciculus ruptured at a different level; microscopic tears, as described in experiment 6
9	Tibial	0.18	15	15.6	..	Same as that in experiment 8
10	Tibial	0.07	37	17.0	..	Longitudinal split in perineurium; pseudoneuroma; complete transverse rupture of all fasciculi at one point and microscopic tears, as described in experiment 6
11	Tibial	0.07	24	29.0	..	Complete transverse rupture of all fasciculi at one point and microscopic tears at various points, as described in experiment 6; perineurium preserved
12	Tibial	0.07	19	32.0	*B*, fig. 3	Complete transverse rupture of individual fasciculi at various levels, as described in experiments 8 and 9; microscopic tears, as described in experiments 8 and 9
13	Tibial	0.20	24	30.0	*D*, fig. 3	Complete transverse ruptures of individual fasciculi at one point and microscopic tears at several levels
14	Tibial	0.20	20	43.0	*A*, fig. 3	Same as described in experiment 13
15	Peroneal	0.13	14.7	27.0	*A* and *B*, fig. 4	Same as described in experiment 13
16	Tibial	0.62	40	?	..	Same as described in experiment 13
17	Ulnar	0.07	14	?	*E* and *F*, fig. 3	Same as described in experiment 13
18	Ulnar	0.07	13	?	*C*, fig. 3	Same as described in experiment 13
19	Ulnar	0.07	4.7	15.3	..	Preservation of perineurium; transverse rupture of myelinated fibers of two larger fasciculi; one fasciculus intact; microscopic tears of individual myelinated fibers and fiber groups at various points throughout segment; "edematous" spaces and irregular spread of endoneurial tissue within ruptured areas
20	Ulnar	0.07	8.5	75.0	..	Transverse ruptures of myelinated fibers in one of two fasciculi and continuity of other, but microscopic tears throughout the nerve segment
21	Peroneal	0.13	9.7	15.0	..	Complete transverse rupture at one point and microscopic tears at several levels
22	Peroneal	0.13	6.2	16.0	..	Transverse tears in various fasciculi at various levels, with preservation of perineurium and larger endoneurial sheaths; evidence of microscopic damage at various levels, as described in preceding experiments

These figures appear to be a satisfactory approximation, in view of the fact that in an ulnar nerve stretched 4.7 per cent under a tension of 15.3 Kg. per square centimeter (nerve 19, table 2) two of the three main fasciculi composing the nerve were completely ruptured, while the third showed evidence of only minor damages at different levels (similar to *F*, fig. 3). Slightly severer histologic damage resulted from stretching to 6.2 and 8.5 per cent, whereas in elongations of from 9.7 to 20 per cent the histologic damage remained almost the same. The average maximal tension at which the greater part of the parenchyma ruptured microscopically amounted to about 27.6 Kg. per square centimeter for 15 ulnar, peroneal and tibial nerves, whereas it was 89.3 Kg. per square centimeter for 2 sciatic nerves. On the other hand, further analysis suggests that the actual tension, expressed in kilograms per square centimeter, is not as good a guide to nerve damage as the actual percentage of stretch. For example, nerve 2 (table 2), which was stretched 10 per cent, showed the same degree of damage as nerve 22, which was stretched 6.2 per cent; yet the tension in the latter was more than five times as great as in the former.

HISTOPATHOLOGIC CHANGES IN NERVE UNDER VARIOUS DEGREES OF STRESS

The macroscopic and microscopic alterations in a stretched nerve involved various structures, depending on the degree of stretching. The perineurium [13a] proved the most resistant structure and withstood overstretching in all our experiments; this means that no experiment was pushed to the point of complete rupture of the perineurium. The greatest elongation in these experiments, 40 per cent in the tibial nerve (table 2, nerve 16), did not result in transverse tears but produced longitudinal splits of the perineurium and herniation of nerve fibers. This type of lesion was observed in nerves stretched as little as 22 per cent. It is probably due to the greater elasticity of the perineurium and the plasticity of the overstretched nerve fibers, which herniated through the opening in the nerve sheath, forming conspicuous bulges. Such pseudoneuromas were observed and described by Denny-Brown and Doherty in the living nerve after the nerve was stretched 100 per cent of its length or more. Herniation began to appear in our experiments after stretching of the sciatic nerve by 21 per cent (table 2, nerve 5) and of the peroneal nerve by 16 per cent (table 2, nerve 3).

The endoneurium appeared to be far less elastic than the perineurium and often tore transversely, together with the nerve fiber bundles, though

13a. The terminology used in the following discussion is adopted from A. A. Maximow and W. Bloom (A Textbook of Histology, W. B. Souonders Company, 1940, p. 190). The epineurium, in its function as the protective cover of the perineurium, is not mentioned separately from the latter in our discussion.

Figure 3

(See legend on opposite page)

it was, as a rule, more resistant than these. Tears of the endoneurium were seen after stretching of about 20 per cent. Below that level, the endoneurium resisted the tension and remained visible as a fine tube around the ruptured nerve fibers. Its connective tissue was pushed into the cavities formed by the microscopic tears of the nerve fibers. The larger endoneurial sheaths carry blood vessels. Microscopic hemorrhages were frequently noted about the endoneurial vessels, whereas larger hemorrhages seemed to occur more frequently in the perineurium.

All nerve fibers were observed to be completely interrupted and retracted within their perineurial tube when the nerve was stretched beyond 10 per cent. The histologic pictures of such a lesion in a tibial nerve stretched 19 per cent is shown in figure 3 *B*. The microscopic appearance of a complete neurofibrillary rupture was rather uniform. The nerve fibers and bundles were torn apart, and the diameter of the nerve was correspondingly decreased. Examination of the remaining part of the nerve revealed that complete rupture of all its nerve fibers was reached through several stages of microscopic damage, in which single nerve fibers or smaller bundles were damaged at various levels.

The histologic picture of the rupture of a single bundle of nerve fibers duplicated that of the rupture of a whole nerve. The fiber bundles which retained their continuity were thinned out and sometimes showed minimal tears. Severance of a whole bundle of nerve fibers likewise

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Fig. 3.—*A*, tibial nerve stretched 20 per cent (experiment 14), showing large tear with discontinuity of most of the myelinated nerve fibers and their endoneurial sheaths. Small microscopic tears of individual fibers appear at other points. Note the fragmentation, globular swelling and poor staining reaction of the myelin near tears.

B, tibial nerve stretched 19 per cent (experiment 12). showing complete rupture of all myelinated nerve fibers and retraction of fibers from the area of the tear. The perineurium is preserved. The nerve shows pronounced thinning.

C, ulnar nerve stretched 13 per cent (experiment 18), showing complete rupture of a large number of nerve fibers, including their myelin sheaths and the endoneurium. There is conspicuous fragmentation of myelin sheaths on both sides of the tear. Some endoneurial tissue is located within the tear. At the right, there is rupture of myelinated nerve fibers but preservation of endoneurial tubes. Microscopic tears of several individual fibers are seen in the lower right corner.

D, tibial nerve stretched 24 per cent (experiment 13), showing an area of microscopic tears of myelinated nerve fibers with preservation of endoneurial tubes. A partly ruptured blood vessel with hemorrhages into the surrounding tissue lies at the right.

E, ulnar nerve stretched 14 per cent (experiment 17), showing small microscopic tears of myelinated nerve fibers with preservation of the endoneurium, which is, however, partly torn. A larger perineurial septum with ruptured vessel and hemorrhage lies at the left. Irregular masses of endoneurium are observed within the tear of the nerve fibers.

F, same nerve as that in *E*, showing area of microscopic tears with preservation of a larger number of nerve fibers. Endoneurial sheaths are preserved within the tears. There is no change in the diameter of the nerve.

leads to decreased circumference of the nerve at that level. The complete tear of a nerve fascicle at one level with preservation of the remaining bundles occurred on stretching a nerve more than 6 per cent of its length.

Of greatest interest were the individual microscopic tears which preceded the interruption of a whole bundle. Examination of a stretched nerve in its entire length revealed that single nerve fibers had been torn haphazardly here and there before rupture of a bundle occurred at one specific point. If only a few nerve fibers had lost their continuity, the fiber bundles might preserve their pattern. Areas of damage were indicated by empty spaces, which appeared translucent in preparations for myelin. They were filled with a fine network springing from the endoneurial system and its histiocytes (fig. 3 *E*). There can be little doubt that these crevices contained fluid in the living nerve and would have suggested the presence of edema of the nerve to the observer. The loose ends of the interrupted nerve fibers seemed to have floated in this liquefied area. Far reaching consequences might be conjectured from the fact that the endoneurium, which had resisted overstretching to this point, had squeezed itself into the space between the fiber stumps (fig. 3 *D* and *E*). In the living nerve, this interfibrillary connective tissue is likely to produce considerable scar formation and to prevent the regenerating nerve fibrils from reaching their goal. Several blood vessels seen in these pictures were torn, and blood corpuscles had entered the perivascular space.

Some insight into the finer mechanism of this lesion was gained by studying the individual nerve fibers under higher magnification. In the right upper quadrant of figure 3 *A* a large tear is seen, accompanied with smaller ruptures of nerve fibers at various other places. The myelin sheaths in the neighborhood of the rupture showed a high degree of fragmentation; those farther away from the tear appeared more uniform, and their staining reaction was better preserved.

These changes are well known to the neuropathologist as globular swelling, fragmentation and degeneration. If observed without knowledge of their origin, they would have to be interpreted as evidence of an intravital pathologic process of at least several days' duration. It was of interest to see that purely mechanical pressure and stress exercised on a dead nerve changed its myelin cylinders in a very similar manner. This observation is borne out by examination of the neurofibrils under oil, as seen in figure 4 *A* to *D*. Figure 4 *A*, from a peroneal nerve stretched 14.7 per cent of its length, shows that the axis-cylinders in an area between two tears are moderately well preserved and the Schmidt-Lantermann incisures fairly normal, especially in the middle of the field. Figure 4 *B* illustrates the same nerve close to the point of its

fixation in the clamp. Here, the myelin was smashed and its pattern destroyed. In figure 4 *C* and *D* are shown additional examples of the effect of pressure and stress on myelinated fibers in areas not directly damaged by rupture. In some nerve fibers, the Schmidt-Lantermann incisures were arranged in a regular fashion; in others the cylindro-conical segments (which are the areas between two incisures) were lengthened, and the incisures themselves appeared widened, as had been observed by Witkowski as far back as 1881.

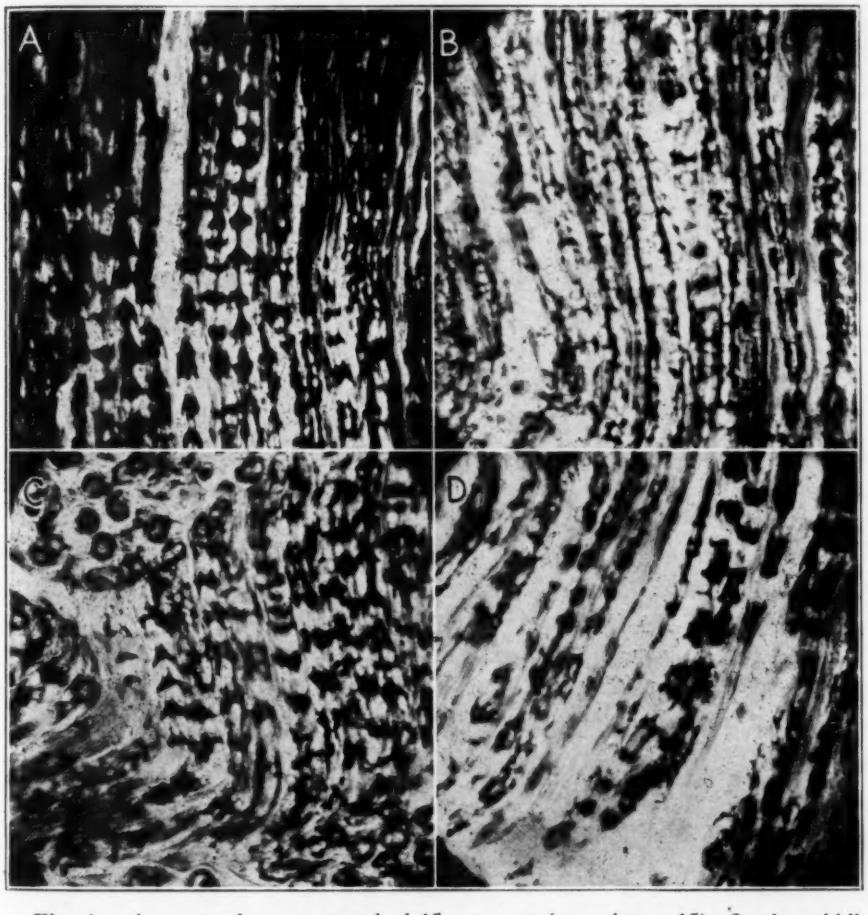

Fig. 4.—*A*, peroneal nerve stretched 15 per cent (experiment 15). In the middle are myelinated nerve fibers with fairly equally spaced "funnels," but the size of the funnels is moderately enlarged, indicating possible widening of Schmidt-Lantermann incisures. Some myelinated fibers are severely damaged, especially at the left.

B, same nerve as that shown in *A*, showing an area of heavier damage to the myelin sheaths with fragmentation, formation of globules and microscopic tears of myelinated nerve fibers.

C, overstretched nerve, showing wavy apeparance with microscopic tears, widening of the Schmidt-Lantermann incisures and discontinuity of the axis-cylinders.

D, result of overstretching on individual myelinated fibers and their staining reaction. Note the fragmentation of myelin, ball formation and discontinuity of myelinated fibers with preservation of endoneurial sheaths.

This observation may give food for thought to the surgeon, who may otherwise be satisfied in having found "the" damage and overlooked the possibility that other parts of the nerve may be involved at the same time. Although recovery without impairment of function seems to be complete in animal experiments, even after much stronger tension, the overstretched nerve is certainly more vulnerable than the undamaged nerve, and this may have some bearing on later surgical intervention.

COMMENT

The tensile strength, as measured by the older authors, was the stress under which the entire nerve ruptured visibly to the naked eye. Tillaux [11] found it to be 20 to 25 Kg. for the median and ulnar nerves. This means 357 Kg. per square centimeter if we use our own value of 0.07 sq. cm. for the cross-sectional area of the ulnar nerve. Trombetta [14] reported 38 Kg., or 543 Kg. per square centimeter. These figures are in agreement with those of Treitel,[13] who found the breaking stress of a tendon of the chicken leg at 459 Kg. per square centimeter, considering that a nerve consists up to about 60 per cent of connective tissue (Ellison [15]).

Our interest, as previously mentioned, is concentrated on the lower end of the stress-strain curve. Denny-Brown and Doherty [10] showed that the first, and full, reversible changes in the living peroneal nerve of the cat are characterized by the transition from the elastic to the plastic state. This was manifested grossly by the nerve remaining extended after being elongated, and microscopically by the beading of the axis-cylinders and the overstretching of the myelin. Are these the changes which occurred at the point where our stress-strain curves became discontinuous? We believe that this was not the case. We have seen that in a nerve extended 4.5 per cent of its length two thirds of all nerve fibers had ruptured, whereas its curve had become discontinuous at a stretch of 3.1 per cent. It is our opinion that the limit of elasticity and the transition to plasticity of the nerve elements are located in the uncharted zone between 0 and 1 per cent strain. This strengthens our assumption that the breaking point of the stress-strain curves actually indicates the danger point beyond which a greater number of nerve fibers snap.

Which parts of a nerve are least resistant to traction and which most resistant? It seems to be the consensus among most investigators that axis-cylinder and myelin are least resistant—be it to direct traction

14. Trombetta, F.: Sullo stiramento dei nervi, Messina, frat. d'Angelo, 1880.

15. Ellison, F. O.: On the Relation Between the Physical, Chemical and Electrical Properties of the Nerves: II. The Tissues Composing a Nerve Trunk, J. Physiol. **39**:397, 1909-1910.

or in consequence of ischemia. Blood vessels, Schwann tubules, endoneurium and perineurium are much more resistant.

Complete interruption of all nerve fibers occurred in our own experiments with 10 per cent stretching of the nerve, whereas with 6 per cent elongation one nerve fascicle may snap with preservation of the other bundles. However, single fibers were found to be torn at various levels of the nerve even at an earlier stage of extension. These histologic observations are in agreement with the results of our stress-strain experiments and curves.

No transverse tear of the perineurium was microscopically observed in this investigation with extension of the nerve to 40 per cent of its total length. A longitudinal split of the perneurial sheath with herniation of nerve fibers occurred on stretching of nerves from 22.4 to 30 per cent. The endoneurium was observed to be torn after about a 20 per cent extension of the nerve.

Thus, it is logical to assume that the distal stump of a severed nerve, consisting of perineurium, endoneurium and Schwann's system only, is at least not less, and is possibly more, resistant than the central one. It may be worth stressing this point, since in an end to end suture of a nerve only the central stump is structurally comparable to the stumps used in our experiment. Degenerated human nerves were not available for stretch experiments.

We come, therefore, to the conclusion, based on our physical and histologic experiments, that no human peripheral nerve must be stretched more than 6 per cent of its mobilized length. Actually, gaps up to 13.5 cm. have been bridged by end to end suture in human ulnar nerves, with excellent functional recovery. We are inclined to see the solution of the seeming contradiction in Highet and Sanders' observation that only 25 per cent of the true elongation of a nerve is due to stretch and 75 per cent to straightening of the mobilized nerve. This question will be discussed in a subsequent clinical paper.[16]

Although the neuropathologic aspect of nerve stretching was not the main item of this study, the observations rendered some insight into the effect of stretching on the human peripheral nerve which was not available from clinical or experimental observations alone. Examination of the whole nerve segment which was exposed to stretching showed the large number of microscopic tears spread throughout the nerve, indicating that the danger zone is evenly distributed throughout the entire elongated area. The damage, therefore, is not restricted to the points where visible tears occur.

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16. Van Wagenen, W. P., and Liu, C. T.: A Clinical Study of Nerve Suture When the Defect Is Large, to be published.

SUMMARY

The necessity of bridging large gaps in injuries to peripheral nerves by means of end to end sutures made it important to obtain reliable data concerning the tensile strength of human peripheral nerves. Experiments in this direction were carried out on human peroneal, ulnar, sciatic and tibial nerves removed at autopsy and stretched with the help of a commercial tensiometer under well controlled conditions. The apparatus and its applications are described. The elongation of nerves under varying degrees of tension was measured.

Stress-strain curves of the data so obtained showed that the point where the curves became discontinuous was located at a mean elongation of 4.2 per cent for ulnar and peroneal nerves. The range of overstretching with which the first minimal damage to nerve fibers appears was found between 3.2 and 5.2 per cent, with a mean of 4.2 per cent of the total length of the nerve.

Histologic examination of 22 overstretched nerves corroborated these observations. The perineurium was observed to split longitudinally, with subsequent herniation of the nerve fibers and formation of pseudoneuromas under a strain of about 22 per cent of the length of the nerve. Transverse tears of nerve fibers and endoneurium begin to appear with an elongation of 20 per cent.

Tears of all myelinated nerve fibers at one point were observed with a stretch of 10 per cent. Individual fasciculi ruptured at different points with a stretch of 6 per cent, and minimal tears of single nerve fibers or small groups at different points of the stretched nerve may occur with an extension as low as 4.7 per cent.

The histologic picture of damaged myelin sheaths produced in our experiments by means of compressing and overstretching an excised nerve appeared almost identical to that of an intravital pathologic process.

A human nerve must not be "stretched" more than about 6 per cent of its mobilized length. The remaining 14 per cent needed to bridge a gap of 13.5 cm. in the ulnar nerve is probably gained by straightening.

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CAUSES OF FAILURE IN TREATMENT WITH ELECTRIC SHOCK

Analysis of Thirty-Eight Cases

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THE INCENTIVE for this article came from a previous report by one of us (N. K. R.) on 200 private patients treated with electric shock and subcoma doses of insulin.[1] The recovery rate of 81 per cent had been maintained from one to five years. Certain factors were stressed as the reason for this high incidence of recovery. Treatment did not stop with shock therapy, but, wherever possible, a follow-up program of psychotherapy, which included not only the care of the patient but a study of the interrelationships of the patient and his immediate family, his school, his work and his social outlets, was insisted on. In practically every case the family was interviewed and made to see its responsibility in the treatment of the patient's illness. In many instances the relatives had severe emotional conflicts, which were corrected. Sociologic factors, wherever possible, were changed and bettered.

No attempt was made to classify these patients on the basis of diagnosis when first seen except to analyze the acute presenting symptoms. A psychiatric diagnosis was attempted later after proper study of the patient, and when he was more accessible to psychotherapy. It is surprising how many mixed psychoses were seen, and how often, with an unbiased approach, an early impression was altered. It was for this reason that a more flexible approach prevailed in the first few interviews and in treatment. This method imposed no rigid standards of classification requiring the treatment to follow a preconceived diagnosis.

Shock therapy was utilized when the patient, owing to agitation, depression or withdrawal, was not accessible to psychotherapy, was unable to take a useful part in his environment and was, by his actions and ideas, dangerous to himself or to others.

From the Psychiatric Center of Seattle.

1. Rickles, N. K.: Electroshock Therapy: A Survey of Two Hundred Cases Treated Over 1 to 5 Year Period in Private Sanatorium, Am. J. Psychiat. **104**:52-55 (July) 1947.

Shock treatment usually consisted of ten or twelve electroshocks. Patients demonstrating schizophrenic features were usually treated with combined courses of electroshock and twenty-five to forty subcoma insulin shocks. The treatment program was considered to have failed if after one or more courses of shock treatment the patient was unable to resume his former activities, to make a contribution to the work in the home and to feel that he could effectively belong to a social unit and could enjoy relationships with that unit. These capabilities should be maintained for at least one year.

In the series of 200 patients there were 38 (19 per cent) who did not respond successfully to shock treatment. In this paper we have attempted to evaluate the possible causes of failure in these 38 patients.

Review of the literature to ascertain the established thought concerning the causes of failure revealed little. However, there were a great deal of conjecture and incomplete prognostic conclusions concerning the patients who had recovered. The general impression gained from reading these articles was one of pessimism. There was a general agreement in regard to certain features which made for recovery, such as a previous good adjustment in school and work, a relatively short period of sickness in a person with an extrovertive makeup, no previous history of mental disturbances and the presence of exogenic precipitating causes. Gottlieb and Hope [2] stated that patients with a warm affect, with normal association and insight, demonstrated under sodium amytal narcosis, were always likely to recover. Kant [3] stressed the importance of extroversion as a predominating personality feature in the course of recovery. Gildea and Man [4] showed that a high lipid content of the blood is synonymous with an extroverted personality and a high rate of recovery. Probably the greatest number of patients with such a background were seen among otherwise normal personalities during the stress and strain of the world war.[5] With these features, it is obvious why the patients made a recovery. It is unfortunate that this happy combination rarely presents itself in civilian practice.

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2. Gottlieb, J. S., and Hope, J. M.: Prognostic Value of Intravenous Administration of Sodium Amytal in Cases of Schizophrenia, Arch. Neurol. & Psychiat. **46**:86-100 (July) 1941.

3. Kant, O.: Evaluation of Prognostic Criteria in Schizophrenia, J. Nerv. & Ment. Dis. **100**:598-605 (Dec.) 1944.

4. Gildea, E. F., and Man, E. B.: Methods of Estimating Capacity for Recovery in Patients with Manic-Depressive and Schizophrenic Psychoses, Am. J. Psychiat. **99**:496-506 (Jan.) 1943.

5. Gold, L., and Chiarello, C. J.: Prognostic Value of Clinical Findings in Cases Treated with Electric Shock, J. Nerv. & Ment. Dis. **100**:577-583 (Dec.) 1944. Rennie, T. A. C.: Analysis of One Hundred Cases of Schizophrenia with Recovery, Arch. Neurol. & Psychiat. **46**:197-229 (Aug.) 1941.

Our experiences with the group of therapeutic failures revealed certain features which indicate a poor prognosis. We were able also to make definite deductions as to the indications for shock therapy and to evaluate more adequately the importance of external factors in the ultimate recovery of the patient. By far the largest number of failures were among the patients with schizophrenic psychoses who had history of insidious onset, gradual withdrawal, disharmony of affect, a feeling of being different and inability to feel deeply or to express oneself easily or freely. The presence of conflict over early sexual acts, such as masturbation, homosexual practices or abortion, was always of poor prognostic significance in these schizophrenic illnesses.

A certain number of patients in the menopausal or postmenopausal period who presented psychoses characterized chiefly by agitated depression would show improvement but then relapse shortly thereafter. The conflict between their religious philosophy and the acceptance of psychiatric help in their basic problems prevented complete recovery. These patients were usually persons who, prior to onset of the psychosis, unsuccessfully sought to find adjustment by identifying themselves with a succession of religious cults, such as Christian Science.

In the cases of severe anxiety neurosis of long standing, on which a psychotic depression had been superimposed, shock therapy usually relieved the depression, but the basic insecurity persisted. In this group of patients insomnia, especially, proved to be the most stubborn of symptoms. Frequently this symptom was relieved during the course of shock therapy but recurred shortly after the cessation of treatment. Such patients have often been seen over a period of years by various physicians. They consult the psychiatrist in the involutional period, a time when their neurotic complaints no longer satisfy their ego. In our experience, shock therapy was often followed by an intensification of the somatic complaints. A certain percentage of the patients in this group who failed to respond to shock treatment had certain types of organic disease, such as hypertension. It is quite possible that the insomnia was due in part to the increased blood pressure. These patients showed no other evidence, however, of memory defects or changes of personality which would be associated with cerebral arteriosclerosis.

We have found that the postpartum psychoses per se usually fail to respond to shock therapy, and it is our contention that the psychosis is usually of toxic origin. We feel that eventually research will result in the isolation of such toxin or toxins.

The most distressing failures occurred in patients who actually had no desire to go on living. Such a reaction was seen typically in the patient who was 45 years of age, or older, and whose husband had died at this stage of the patient's life. Such patients were always dangerous as potential suicides.

Many articles in the literature emphasize the belief that a history of previous shock treatment renders the prognosis rather poor. Such has not been our experience, as we have frequently had patients who responded well to a subsequent course or courses of treatment.

We found that many patients who did not have a complete recovery with shock therapy eventually were capable of making a fairly good adjustment in the home, owing in large part to the existence of a friendly environment, in which members of the patient's family were able to demonstrate continued affection and extreme patience. We feel that the psychiatrist often expects too much improvement in the atmosphere of the hospital. In certain cases the proper therapeutic approach is not further shock therapy and continued hospitalization but, rather, the return of the patient to his home, with treatment continued on an ambulatory basis.

It is our impression that electroshock therapy is not indicated in treatment of the manic phase of the manic-depressive psychosis. While some patients seem to improve with shock therapy, they often have a recurrence of the manic phase, and the relapse is characterized by symptoms which are severer than the original ones. A definite paranoid coloring of their thought content is expressed, and it becomes extremely difficult to manage the patient. This reaction is probably an expression of deeply guilt-laden, repressed material, such as latent homosexuality. The same reaction was seen in the past, before the advent of shock therapy, in patients who had had prolonged periods of manic disturbances and whose psychoses manifested a progressive chronicity.

ILLUSTRATIVE CASE HISTORIES

The following case histories illustrate the preceding statements, and the accompanying tables indicate the clinical findings, the treatment

TABLE 1.—*General Data on 38 Patients for Whom Electroshock Therapy Failed*

Sex		Diagnosis					
Male	Female	Schizophrenia	Manic-Depressive Psychosis	Anxiety Neurosis	Postpartum Psychosis	Psychopathic Personality	Involutional Melancholia
10	28	18	9	6	1	2	2

Marital Status				Age Groups, Yr.				
Single	Married	Widowed	Divorced	20-30	30-40	40-50	50-60	60-70
12	20	3	3	7	15	10	1	5

No. of Acute Breakdowns				Courses of Treatment			
One	Two	Three	Four	First	Second	Third	Fourth
11	17	4	6	15	12	8	3

Present Status				
State Hospital	Home	Sanatorium	Suicide	Death
7	21	6	3	1

given and the present status of 38 patients who failed to recover as a result of shock therapy.

The first case to be presented is illustrative of a schizophrenic illness with gradual onset and a history of conflict over early sexual

TABLE 2.—*Detailed Clinical Data on 38 Patients for Whom Electric Shock Therapy Failed*

Sex*	Age, Yr.	No. Nervous Breakdowns	Marital Status	Therapy: Electric Shock	Therapy: Subcoma Insulin	Present Situation
				Schizophrenia		
F	41	2	S	3	2	State hospital, deteriorated
F	37	2	M	3	2	Good social adjustment; home
F	23	2	S	3	3	Private sanatorium, suspicious, deteriorated
M	35	3	S	3	1	Home; fair adjustment
F	35	2	M	2	2	State hospital, deteriorated
F	23	3	S	3	3	Private sanatorium, bizarre behavior
F	42	3	M	2	1	Suicide in county hospital
M	41	2	S	2	1	Home; poor adjustment, bizarre behavior
F	34	2	S	3	3	State hospital, deteriorated
F	32	2	M	2	..	Good adjustment, home, 1 yr. p Rx.
F	37	1	M	2	..	Home; good adjustment
F	33	2	M	1	..	State hospital; attempted suicide
F	39	1	M	..	1	State hospital
F	31	2	D	1	1	Suicide at home
F	26	1	M	1	1	Suicide at home
M	42	1	S	1	1	State hospital
F	33	2	S	1	1	Home; paranoid, delusional and hallucinated
F	44	1	W	1	1	Home: poor adjustment, suspicious
				Manic-Depressive Psychosis		
F	32	4	S	1	..	Private sanatorium, in manic phase
F	31	3	M	2	..	Home; fair social adjustment, suspicious
M	29	2	M	2	..	Home; improving
F	23	4	S	1	..	Private sanatorium; depressed, improving
F	49	4	M	2	..	Home; good social adjustment
F	39	4	D	4	..	Home; poor adjustment, paranoid
M	41	4	M	3	..	Home; good adjustment, 1 yr. p Rx.
F	35	1	M	1	..	Home; good adjustment
F	65	2	W	3	..	Private sanatorium; depressed, deteriorated
				Anxiety Neuroses with Depression		
M	65	2	M	4	..	Died, cerebral hemorrhage, 5 mo. p Rx.
F	67	2	M	2	..	Home; good adjustment, insomnia
F	68	1	M	1	..	Home; good adjustment, gastrointestinal complication
F	47	4	M	4	..	Sanatorium; acute agitated depression
F	43	2	M	1	..	Home; fair adjustment
F	27	1	S	1	..	State hospital; attempted suicide
				Postpartum Psychoses		
F	28	1	M	1	..	Home; gradual improvement
				Psychopathic Personalities with Depression		
M	36	2	D	2	..	Home; poor social adjustment, alcoholic
M	47	2	S	2	..	Home; good adjustment, moderately alcoholic
				Involutional Melancholias		
M	65	1	M	1	..	Home; good social adjustment
M	51	1	W	2	..	Home; good social adjustment

* Males, 10; females, 28.

experiences. Of the patients who failed to respond to treatment, the largest percentage were in the schizophrenic category.

CASE 1.—Mrs. J. S., a woman aged 32, had been married for six years and had 2 children. When she first came to our attention, in January 1946, her husband took three hours to tell us that his wife had been suspicious, apathetic and irritable

for at least a year and had completely neglected the children. It was also brought out that she spoke to none of the neighbors, and that she refused to let her husband use contraceptives and yet dreamed of hurting her children. She was a self-conscious child who was backward and said little. She failed in nurse's training, and her superiors felt that they were unable to ascertain how much she actually knew. She had an unhappy love affair when she was about 26, and in a depressive state she had sexual relations with her brother. Her mother was a selfish, irritable, unpleasant person. Her father had an anxiety neurosis. Her husband felt that the family knew that the patient was mentally ill at the time of her marriage but kept quiet so that they could get rid of her.

The patient received a combined course of subcoma insulin shock and electroshock therapy in the hospital from Jan. 18 to March 11, 1946. No real rapport developed between the physician and the patient, and she acquired no real insight; however, she became cheerful and rather pleasant and took considerable interest in her personal appearance. She remained fairly well for two weeks after her return home and then completely reverted to her original status. The husband reprimanded her quite openly for wanting to remain in bed the second morning she was home and felt that she should have prepared his breakfast for him. He felt that he wished to risk no further money on her medical care and that her presence in the home was extremely harmful to the children. Accordingly, the patient was committed to a state hospital. There is ample evidence that the patient was rejected both by her own family and by her husband. We feel that the failure in treatment was due partly to the fact that the home environment lacked a healthy member. Unless the home environment is one that makes the patient feel secure, the difference between the home and the hospital will be so obvious that the patient will be quite apt to fail of cure.

Case 2.—Mrs. J. R. represents the group of patients who have, over a period of years, been ardent followers of a certain faith and who become depressed and confused when thoughts of previous sex practices recur. Her conflict arose because she could not reconcile her past conduct with her present religious beliefs. She was a woman of 65, short and stockily built, and had raised 4 children. Her sickness followed the death of her husband four years before and has persisted to date. She objects to any medication or shock treatment because of the teachings of her faith but does show definite temporary improvement after shock therapy. This patient is not accessible to psychotherapy, owing to her age and her profound religious beliefs.

Case 3.—J. N., a man aged 63, had had a long-standing psychoneurosis and became ill with a severe depression in the involutional period. The case illustrates that electroshock therapy will relieve a great deal of the depression of such patients, but that insomnia will often recur and somatic complaints become more evident as the depression is alleviated.

Because of severe depression, the patient had had four courses of electroshock over a period of a year and a half, the longest series consisting of ten treatments. His depression would respond to shock therapy for about three months and then there was a gradual return to the insomnia, anxiety and somatic complaints referable to the heart and gastrointestinal tract. The patient had been an only child; his background and education were excellent. He was a graduate attorney; yet he had been satisfied for over thirty-five years to remain at a desk job doing routine legal work. His justification for this was the state of his health; he had always been afraid to assume any new responsibility. He had seen many physicians during the course of the century, and diagnoses of colitis, cardiac neurosis, migraine and hypertension had been made. Physical examination showed

moderately severe hypertension. In the latter part of 1946 hypertensive retinopathy developed, with hemorrhages in the right eye. Owing to his age and the chronicity of his complaints, it was felt that deep psychotherapy would not be effective.

His depression started with the fear of competition from younger attorneys and some discord in the office in which he worked. He had made a mild attempt at suicide on at least two occasions.

We feel that this man had benefited from shock therapy, inasmuch as the depression cleared up with each course of treatment. However, owing to his basic personality makeup, with persistent insomnia and anxiety, the case must be classified as one of failure from the standpoint of complete recovery. His last course of electroshock treatments was given in November 1946. He was able to return to work in January 1947 and continued up to April 24, when he had a cerebral hemorrhage and died the following day, five months after the last course of electroshocks. It is surprising that this man was able to undergo four courses of electric shock with no outward physical effect at the time, in spite of hypertension with a systolic pressure of from 170 to 200. He at no time demonstrated any memory defects.

CASE 4.—Mrs. B. L., aged 28, prior to her present illness had led a normal and useful life. The onset of her mental disturbance was about the third day after delivery of a normal boy. She became confused and disoriented, had many bizarre hallucinations and delusions, refused to eat and became generally uncooperative. She was given a combined course of twenty electroshocks and eight weeks of insulin subshock therapy. Her condition remained unchanged during the course of treatment. Finally, after twelve weeks of care in the sanatorium, she was discharged to her home. At present, six months after her discharge, she is beginning to make a fair adjustment and to show some interest in her child and family. Practically all her delusions have disappeared. It is our impression that this patient would have made the same recovery without shock therapy and that her illness was undoubtedly due to a toxic state rather than to a functional condition.

CASE 5.—This patient is representative of those who do not wish to go on living and whose illness has come on in the involutional period. The psychosis is precipitated by the loss of the marital partner. The personality is one of long-standing inadequacy.

Mrs. D. W., aged 43, was examined in April 1946. Her illness began after her husband's death, in September 1944. She became depressed, irritable and indecisive. Her financial circumstances demanded that she sell her home. Her sister described the patient as an extremely religious, socially inadequate, overly irritable person, who never got along with her husband, her neighbors, the school authorities or her two sisters. Her mother had been in and out of sanatoriums for the last twenty years because of repeated episodes of paranoid depression. She had lived with the patient since the death of the patient's husband. The patient's youngest sister had spent most of her earnings on her mother and given most of her spare time to the patient. The patient was admitted to the hospital for a combined course of subcoma insulin treatments and electroshock therapy on April 23, 1946 and was discharged on May 14. Her decision to leave early was influenced by the cost of the hospital care. Her improvement in the hospital was comparable to that in case 1. She relapsed completely within three weeks after leaving the hospital and attempted suicide.

She had rejected the sale of her house because she was unable to get her price for it. The death of her husband made her financially insecure and forced

her to make decisions which she had never needed to make before in her life. The younger sister, who had supported the patient emotionally for many years, finally became tired and worn out and was unable to continue to make her contribution.

CASE 6.—Mrs. J. E., aged 23, after disappointment in a love affair, took 15 grains (0.975 Gm.) of pentobarbital sodium, with the intention of suicide. She was placed in a sanatorium and given shock therapy, but her apathy and disinterest continued. Care in a state hospital was recommended. We were called in consultation and stated the belief that this patient could probably make a fair adjustment with another course of treatments. She was transferred to our care and given twelve electroshock treatments. She responded well and at present, one year after treatment, has made a splendid adjustment and is completely normal. This case is discussed to demonstrate that previous treatment does not necessarily mean a poor prognosis.

Two cases are presented to emphasize that some patients do not show a great deal of improvement in the hospital after shock treatment, but that after their return home a fairly good adjustment is eventually secured and maintained.

CASE 7.—Mrs. B. W., aged 33, was first examined on Oct. 28, 1944. At that time a history was obtained that in 1934, the year of her marriage, she became depressed and suicidal and was hospitalized for six months. Her depression recurred early in October 1944, three weeks after she was delivered of a baby girl. Her 5 year old son had died of leukemia in 1942, and the patient and her husband were both disappointed because their newborn child was not a boy. On examination in October 1944 she was uncooperative and resistive. She had feelings of guilt over masturbation and over her son's death. She felt that her parents-in-law rejected her because she was not overly religious. She had always been a shy, modest person, who had taken life seriously. She had been frigid sexually. Her menses had been irregular. She was born in Poland and was brought to the United States when 3 years old. She completed high school and had been employed as a bookkeeper prior to her marriage. Her father drank excessively, had had one serious mental breakdown and had never made a good living. Her brother had had a schizophrenic upset. The patient's mother had had hysterical upsets most of her life. Her father-in-law was extremely religious. Her husband was the patient's second cousin. He was a passive, hard-working person, who was extremely devoted to his wife. His sexual life has usually been characterized by premature ejaculation. They have 2 living daughters.

The patient received electroshock therapy in October 1944. She improved very slowly and remained in the hospital until June 1945. She attempted suicide four days later and was readmitted to the hospital for two days, during which time she received two electroshock treatments. She was readmitted to the hospital in August 1945 because of hallucinations, feelings of being influenced and apathy. She received six electroshock treatments and was discharged to her home after three weeks in the hospital. She did poorly at home and was readmitted for the fourth time in sixteen months early in January 1946. During her stay this time she received electroshock and subcoma insulin shock therapy and sodium amytal narcotherapy. Her improvement was slow, and she remained rather apathetic. She was discharged Feb. 15, 1946. While at home she improved, despite the occurrence of two deaths in the family, the illness of one sister and the further fact that in July 1946 she became pregnant. A therapeutic abortion and sterilization

were recommended, and the patient tolerated the procedure fairly well emotionally. She now manages her home efficiently and is rather active socially.

Throughout the patient's long illness her husband has remained patient, cooperative and understanding. We feel that his attitude has been a tremendous help and that all the other measures would have been of no permanent value if her husband had been less effective.

CASE 8.—V. W., a Catholic housewife aged 33, was first examined on April 4, 1946. At that time a history was obtained that she had been extremely depressed since her husband had an attack of coronary thrombosis in July 1945. Actually, she had had a moderate degree of depression for about two years, due to the death of her mother in 1944 and of her brother and father in January 1945. During the course of the illness, the patient had choking spells, anorexia, sadistic urges toward her 2 year old child and visual hallucinations. In 1942 she had a miscarriage and has since then not felt well physically. She had always been overly emotional and unduly concerned about life after death. Her father was depressed after his wife's death in 1944.

The patient received subcoma insulin treatment and electroshock therapy on two separate occasions (May and June 1946). She improved somewhat after the first course and left the hospital comparatively early, owing to the family's financial situation. When examined in the hospital on July 5, after eight electroshock treatments and fifteen subcoma insulin treatments, she was extremely preoccupied with religious thoughts and talked of her mother at great length. Her husband took her out of the hospital, against advice, on July 15. When examined on July 26, 1946, at the office, she was perplexed and was in only partial contact with reality. Contrary to our prognostic expectations, the patient has shown progressive improvement, and at present is doing a fair job caring for her home and child. For all practical purposes she is still at least mildly psychotic; but with her husband's help she is adjusting comparatively well.

CASE 9.—Failure of the manic phase of a manic-depressive psychosis to respond to electroshock is illustrated by this case.

I. W., a man aged 43, had had four attacks of manic-depressive psychosis. He was a graduate of the University of Washington. An only child, he had been greatly pampered as a youngster. He had never been able to make a satisfactory adjustment, although he had a pleasant personality and made friends easily. He was married to a woman older than he, from whom he could feel free from any strong sexual drives and feelings of competition where other men were concerned. His last manic episode began in May 1946. He received electroshock therapy and initially seemed to improve. His manic symptoms then became severer and were associated with paranoid delusions with a large amount of homosexual coloring. He was transferred to a state hospital in June 1946. He made a gradual recovery without shock therapy and was discharged in March 1947. When he was last seen in the office, on April 28, 1947, he was gainfully employed and fairly well adjusted socially.

SUMMARY AND CONCLUSIONS

This report concerns 38 of a series of 200 patients who failed to respond favorably to shock treatment. The course of treatment usually consisted in a series of ten or twelve electroshock treatments. Patients who demonstrated schizophrenic features usually also received twenty-four to forty subcoma insulin shocks. Treatment was considered to

have failed if satisfactory improvement was not maintained for at least one year.

We have learned to be cautious in drawing pessimistic conclusions at a relatively early phase of the process. The great value of ambulatory treatment after the patient has been discharged from the hospital is to be emphasized repeatedly. We have seen patients make unexpected gains in an atmosphere which included understanding relatives. The clinician must learn to be slow in recommending institutional care, especially when the family is willing to assume responsibility in providing adequate home facilities. In cases in which such a provision is economically impossible, it should be the future aim in the treatment of functional psychoses to foster small, homelike state facilities, where a patient will not feel lost and forsaken and where the sustaining members of the family can continue to visit and give affection and hope.

Shock therapy, in our opinion, is apt to fail if the patient has in the past been a suspicious, socially inadequate person, if there is a long-standing psychoneurotic type of response to life situations and if the patient is in the menopause or postmenopausal period and has lost a marital partner on whom she has been extremely dependent. The patient who for many years has sought an adjustment through religious identifications often will fail to respond to shock therapy. Our experience has also demonstrated that the manic patient does not respond favorably to electroshock.

We are aware of the small number of cases in our series and the lack of statistical proof for our deductions; however, we have hoped by this report to stimulate others to make similar, comprehensive analyses of the conflicts in patients who have failed to respond to electroshock treatment and of the environmental factors which may have contributed to this failure. When this is done, conclusions may become available which may enable psychiatrists to be more effective and selective in the use of shock therapy in the future.

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PENICILLIN IN TREATMENT OF NEUROSYPHILIS

IV. Tabes Dorsalis

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AS PART of the nation-wide evaluation of penicillin in the treatment of syphilis, a study of the use of this drug with various forms of neurosyphilis has been in progress at the Johns Hopkins Hospital since October 1943. The preliminary results of therapy of asymptomatic neurosyphilis [1] and dementia paralytica [2] and the early changes in the abnormalities of the cerebrospinal fluid [3] have been reported in previous papers. This study, the fourth in the series, is concerned with the treatment of tabes dorsalis.

The details of therapy and the methods of analyzing the results have been described in earlier publications and need not be repeated here. The same reservations made necessary by the changing character [4] of commercial penicillin during the period of this study are necessary.

As of October 1946, 54 patients with tabes dorsalis have been treated with penicillin. Thirty-three of these patients (group A) received penicillin alone in amounts ranging from 1,200,000 to 20,000,000 units in divided doses; the remaining 21 patients (group B) received from 1,800,000 to 20,000,000 units of penicillin in divided

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From the Venereal Disease Division of the Medical Clinic, Johns Hopkins Hospital, and the United States Public Health Service and Johns Hopkins University Venereal Disease Research and Post-Graduate Training Center.

1. Moore, J. E., and Mohr, C. F.: Penicillin in the Treatment of Neurosyphilis: I. Asymptomatic Neurosyphilis, Am. J. Syph., Gonor. & Ven. Dis. **30:** 405 (Sept.) 1946.

2. Reynolds, F. W.; Mohr, C. F., and Moore, J. E.: Penicillin in the Treatment of Neurosyphilis: II. Dementia Paralytica, J. A. M. A. **131:**1255 (Aug. 17) 1946.

3. Reynolds, F. W.: Penicillin in the Treatment of Neurosyphilis: III. Cerebrospinal Fluid Changes in Cases of Symptomatic Neurosyphilis, Ann. Int. Med. **26:**393 (March) 1947.

4. Committee on Medical Research, the United States Public Health Service and the Food and Drug Administration: The Changing Character of Commercial Penicillin, with Suggestions as to the Use of Penicillin in Syphilis, J. A. M. A. **131:**271 (May 25) 1946.

doses concurrently with induction of malarial fever. Patients with the tabetic form of dementia paralytica were excluded from the present series because of the manifest difficulty in obtaining reliable evaluations of symptoms from patients with the psychoses of this disease.

The results presented in this paper must be interpreted not only in the light of the small number of cases reported and the relatively brief periods of post-treatment observation, but also with due regard to the fact that tabes dorsalis is a chronic and notoriously capricious condition, the manifestations of which often are largely subjective and given to spontaneous remissions. To assess "improvement" following any form of therapy of this condition is often a matter of considerable difficulty; to relate improvement specifically to therapy requires prolonged and meticulous post-treatment observation.

The spontaneous evolution of tabes dorsalis in the absence of treatment is imperfectly understood, and all gradations of severity and rapidity of progress are observed. There are patients for whom the diagnosis can be established on the basis of objective physical signs but who have never had any of the distressing symptoms associated with the disease. In others but one symptom may develop, and this may persist unchanged for decades. In still others the evolution is rapidly progressive to complete invalidism. Spontaneous remissions of varying duration occur, the symptoms recurring unpredictably.[5]

In tabes, as in other forms of neurosyphilis, actual destruction of nerve tissue (e.g., in the posterior columns or the optic nerves) may be responsible for irremediable damage with persistence of both subjective and objective evidence. On the other hand, the pathogenesis of certain important symptoms of tabes (e.g., lightning pains or gastric crises) is incompletely understood and, in the light of present knowledge, cannot be related clearly to permanent pathologic damage to neural structures. This discrepancy between clearly defined clinical entities and definite knowledge of the neuropathologic lesions which produce them makes an evaluation of results of treatment even more difficult than with the less complex forms of neurosyphilis.

Moreover, it should be pointed out that tabes is primarily a disease of subjective symptoms, and therefore one for which there are few objective measures of "improvement," the latter depending largely on the patient's own appraisal of his condition. Into his interpretation enter many psychosomatic factors. How, for example, can a complaint such as impotence accurately be related to neurosyphilis alone? Patients with tabes frequently are of an age in which prostatism occurs. How, then, can one fully evaluate from month to month a complaint of

5. A recrudescence of lightning pains is frequent in the course of any intercurrent febrile illness. We have a patient who has typical gastric crises regularly, and only at the time of menstruation.

hesitancy of urination in a tabetic patient who also has benign prostatic hypertrophy? Indeed, the importance of the obstructive factor in the tabetic bladder due to neurogenic contracture of the vesical neck has only recently been recognized.[6]

These and other considerations have led such careful workers as Dattner, Thomas and Wexler[7] to the conclusion that the efficacy of treatment of neurosyphilis should be adjudged not at all by the clinical symptoms but solely by the response of the cerebrospinal fluid—specifically, by the cell count and determination of the total protein, which they believe indicate "activity" of the process in the central nervous system.

Nevertheless, the patient and his physician are far less interested in the cell count and protein content of the spinal fluid than in knowing the answers to such questions as: "Will this treatment relieve lightning pains?"; "What are the prospects that ability to walk will be improved?" and "Is it possible to ameliorate such symptoms as paresthesias, impotence and incontinence of urine?" Surely, an evaluation of the effects of penicillin in the treatment of tabes dorsalis must encompass a consideration of its effects on the symptoms of the patient to whom it is given!

GROUP A: PATIENTS TREATED WITH PENICILLIN ALONE

Case Material.—Thirty-three patients with tabes dorsalis were treated with penicillin alone. The average age of these patients was 47 years, the youngest being 29 and the oldest 79. The age, race and sex distributions are shown in table 1.

Fifteen patients (45 per cent) had had a comparatively recent onset of symptoms, i. e., within less than two years. Fourteen (42 per cent) had had symptoms for two years or more. The duration of symptoms, the extent of the pretreatment abnormalities of the cerebrospinal fluid and the duration of post-treatment observation are shown in table 2. Twenty-one patients had received previous antisyphilitic therapy: 3, fever therapy and metal chemotherapy; 18, metal chemotherapy alone. In no case had fever therapy been given within two years prior to the institution of penicillin therapy. In none had there been any significant symptomatic improvement from previously administered treatment.

Effects of Therapy.—(*a*) Herxheimer Reactions: Primary febrile reactions occurring within the first thirty-six hours after the institution of penicillin therapy were noted in 4 patients in the present series. The spinal fluids of all 4 patients were of group III, with strongly positive serologic reactions and evidence of "activity."[7] In 2 patients there seemed to be a transitory exacerbation of lightning pains. Neither had a concomitant febrile Herxheimer reaction. There was no correlation between the occurrence of these reactions and the results of therapy.

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6. Emmett, J. L.: Transurethral Resection in Treatment of True and Pseudo Cord Bladder, J. Urol. **53**:545 (April) 1945.

7. Dattner, B.; Thomas, E. W., and Wexler, G.: The Management of Neurosyphilis, New York, Grune & Stratton, Inc., 1944.

It is notable that febrile Herxheimer reactions following penicillin therapy were significantly less frequent among patients with tabes dorsalis (12 per cent) than among the previously reported patients with dementia paralytica (79 per cent).[2]

TABLE 1.—*Age, Race and Sex Distribution of Patients Treated with Penicillin Alone*

Age, Years	Race	Sex		Total Number		
		Male	Female	White	Negro	All Patients
20-29	White	1	0	1	..	2
	Negro	0	1	..	1	
30-39	White	4	1	5	..	9
	Negro	4	0	..	4	
40-49	White	5	3	8	..	11
	Negro	2	1	..	3	
50-59	White	2	0	2	..	5
	Negro	3	0	..	3	
60 (over)	White	5	1	6	..	6
	Negro	0	0	..	0	
Total number		26	7	22	11	33

TABLE 2.—*Duration of Symptoms, Extent of Pretreatment Abnormalities of the Cerebrospinal Fluid and Duration of Post-Treatment Observation in Patients Treated with Penicillin Alone*

Spinal Fluid Group	Duration of Symptoms, Years	Duration of Post-Treatment Observation, Months						Totals
		0-6	6-12	12-18	18-24	24-30	30-36	
Negative	0-2	..	..	..	..	..	..	4 (12%)
	2-4	..	..	..	..	..	..	
	More than 4	..	2	..	..	1	..	
	Unknown	..	..	1	..	..	..	
Group II (Inactive)	0-2	..	..	..	..	..	1	8 (24%)
	2-4	..	..	..	..	2	..	
	More than 4	1	1	..	1	1	..	
	Unknown	..	..	..	..	..	1	
Group II (Active)	0-2	..	3	..	..	1	..	6 (18%)
	2-4	..	..	..	..	1	..	
	More than 4	..	..	1	..	..	..	
	Unknown	..	..	..	..	..	..	
Group III (Strongly active)	0-2	..	4	2	2	2	..	15 (45%)
	2-4	..	1	..	1	..	..	
	More than 4	..	1	..	..	..	..	
	Unknown	..	1	..	..	1	..	
Totals		1	13	4	4	9	2	33

(*b*) Changes in Abnormalities of the Cerebrospinal Fluid: As with asymptomatic neurosyphilis [1] and with other clinical syndromes of syphilis of the central nervous system,[3] the abnormalities of the spinal

fluid associated with tabes dorsalis responded favorably to treatment with penicillin. Elevated cell counts and total protein levels became normal promptly, usually within a few weeks. This was followed by more gradual, but equally well sustained, improvement in the results of colloidal mastic tests and in the titer of the Wassermann test. The effects on the abnormalities of the spinal fluid in the 33 patients treated with penicillin alone are shown in chart 1.

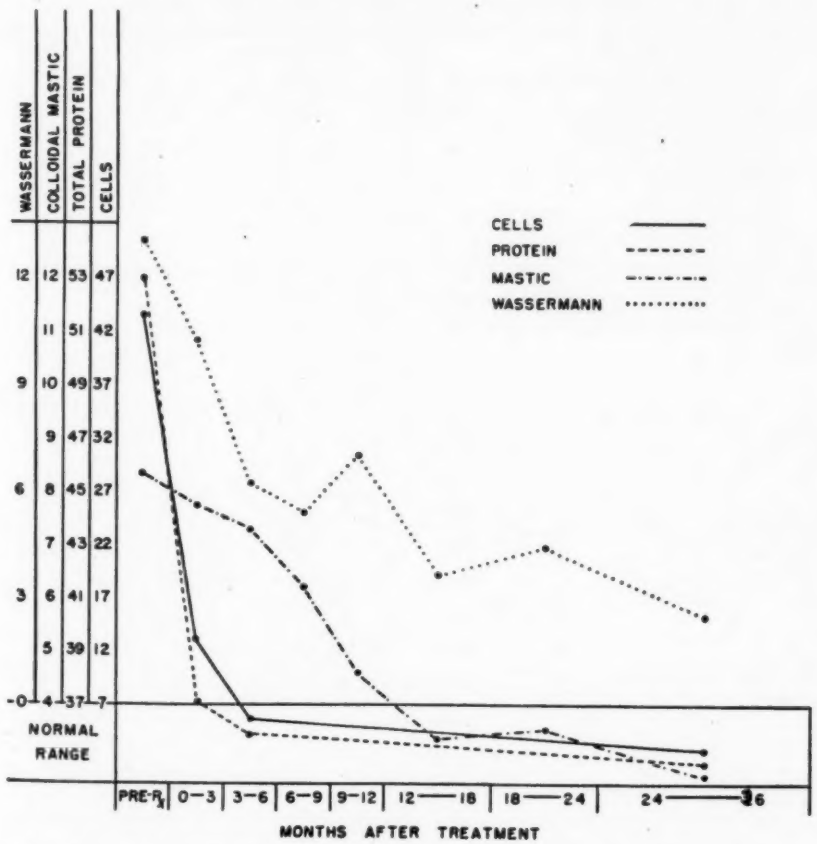

Chart 1.—Effects of therapy with penicillin alone on the abnormalities of the cerebrospinal fluid of 33 patients with tabes dorsalis.

(*c*) Effects on Clinical Manifestations of Tabes Dorsalis: As judged by the general status of the patients at the time of their most recent examination, 9 of the 33 patients (27 per cent) in group A were significantly benefited from therapy with penicillin alone. The condition of 3 others appeared to have been improved temporarily, but subsequently again became as bad as, or worse than, it had been prior to treatment. Eighteen showed essentially no change at the time of the last examination, and the condition of 6 was worse.

The effects of penicillin therapy on the various symptoms of tabes dorsalis are shown in table 3. Here it will be noted that penicillin appears to be most efficacious in affording relief of lightning pains and in ameliorating gastric symptoms.

As might be expected, penicillin had practically no effect on the objective signs of tabes. In 1 patient, deep reflexes in the legs, which had been absent, were present, although hypoactive, after treatment. In another, a partial return of the deep pain sense in the achilles tendons was recorded.

TABLE 3.—*Effects of Penicillin Therapy on Symptoms of Tabes*

Symptom	Number of Cases	Significant Improvement	No Significant Change	Condition Worse
Lightning pains	20	9	9	2
Ataxia	10	2	5	3
Urinary symptoms	11	2	7	2
Sensory disturbances	7	2	3	2
Gastric symptoms	5	3	2	0
Impotence	4	1	3	0
Ocular palsies	1	1	0	0

TABLE 4.—*Age, Race and Sex Distribution of Patients Receiving Combined Malaria and Penicillin Therapy*

Age, Years	Race	Sex		Total Number		
		Male	Female	White	Negro	All Patients
20-29	White	0	0	0	..	0
	Negro	0	0	..	0	
30-39	White	6	2	8	..	9
	Negro	1	0	..	1	
40-49	White	6	3	9	..	9
	Negro	0	0	..	0	
50-59	White	2	0	2	..	2
	Negro	0	0	..	0	
60 (over)	White	0	1	1	..	1
	Negro	0	0	..	0	
Totals		15	6	20	1	21

GROUP B: PATIENTS TREATED WITH MALARIA AND PENICILLIN

Case Material.—Twenty-one patients with tabes dorsalis were treated with malaria and penicillin. The average age of these patients was 42, the youngest being 34 and the oldest 62. The age, race and sex distributions are shown in table 4.

Eight patients (38 per cent) had had symptoms for less than two years, and 11 (52 per cent), for two years or more. The duration of symptoms, the extent of the pretreatment abnormalities of the cerebrospinal fluid and the duration of post-treatment observations are shown in table 5.

Fifteen patients had received previous antisyphilitic therapy: 4, fever therapy and metal chemotherapy; 3, metal chemotherapy and penicillin, and 8, metal chemotherapy alone. To only 1 patient had either fever or penicillin therapy been given within eighteen months prior to the institution of malaria and penicillin therapy. In none had there been any significant symptomatic improvement from previously administered treatment.

TABLE 5.—*Duration of Symptoms, Extent of Pretreatment Abnormalities of the Spinal Fluid and Duration of Post-Treatment Observation for Patients Treated with Malaria and Penicillin*

Spinal Fluid Group	Duration of Symptoms, Years	Duration of Post-Treatment Observation, Months						Totals
		0-6	6-12	12-18	18-24	24-30	30-36	
Negative	0-2	..	..	1	..	..	..	8 (14%)
	2-4	..	..	..	..	..	..	
	More than 4	1	..	1	..	..	..	
	Unknown	..	..	..	..	..	..	
Group II (Inactive)	0-2	..	..	..	..	1	..	4 (19%)
	2-4	..	1	..	..	..	..	
	More than 4	1	..	..	..	..	1	
	Unknown	..	..	..	..	..	..	
Group II (Active)	0-2	1	1	..	..	..	..	3 (14%)
	2-4	..	1	..	..	..	..	
	More than 4	..	..	..	..	..	..	
	Unknown	..	..	..	..	..	..	
Group III	0-2	..	2	..	1	1	..	11 (52%)
	2-4	1	2	..	..	..	..	
	More than 4	..	1	..	..	1	..	
	Unknown	..	..	1	1	..	..	
Totals		4	8	3	2	3	1	21

TABLE 6.—*Effects of Combined Malaria Plus Penicillin Therapy on Symptoms of Tabes Dorsalis*

Symptoms	Number	Significant Improvement	No Significant Change	Condition Worse
Lightning pains	15	13	2	0
Ataxia	11	7	4	0
Urinary symptoms	8	4	4	0
Sensory disturbances	7	3	3	1
Gastric symptoms	4	4	0	0
Impotence	6	2	4	0
Cranial nerve palsies	1	1	0	0

Effects of Therapy.—(*a*) Herxheimer Effects: Febrile Herxheimer reactions could not be detected in any of the patients in this group, since penicillin was given concurrently with the malarial treatment. Ten of 15 patients with lightning pains had exacerbation of this pain during the first few days of therapy; almost invariably, this was most apparent at the height of the malarial paroxysm. Whether this phenomenon should be classified as a Herxheimer reaction is doubtful, since it has been observed long after treatment was concluded (e. g., with fever due to acute infections of the respiratory or urinary tract).

(*b*) Changes in Abnormalities of the Cerebrospinal Fluid: The changes in the abnormalities of the spinal fluid following concurrent malaria and penicillin therapy are shown graphically in chart 2. As with the patients treated with penicillin alone, the increased cell counts and elevated protein levels of the spinal fluid promptly returned to normal. Improvements in results of colloidal mastic tests and in titers of the Wassermann test were more dramatically clearcut than those following the use of penicillin alone.

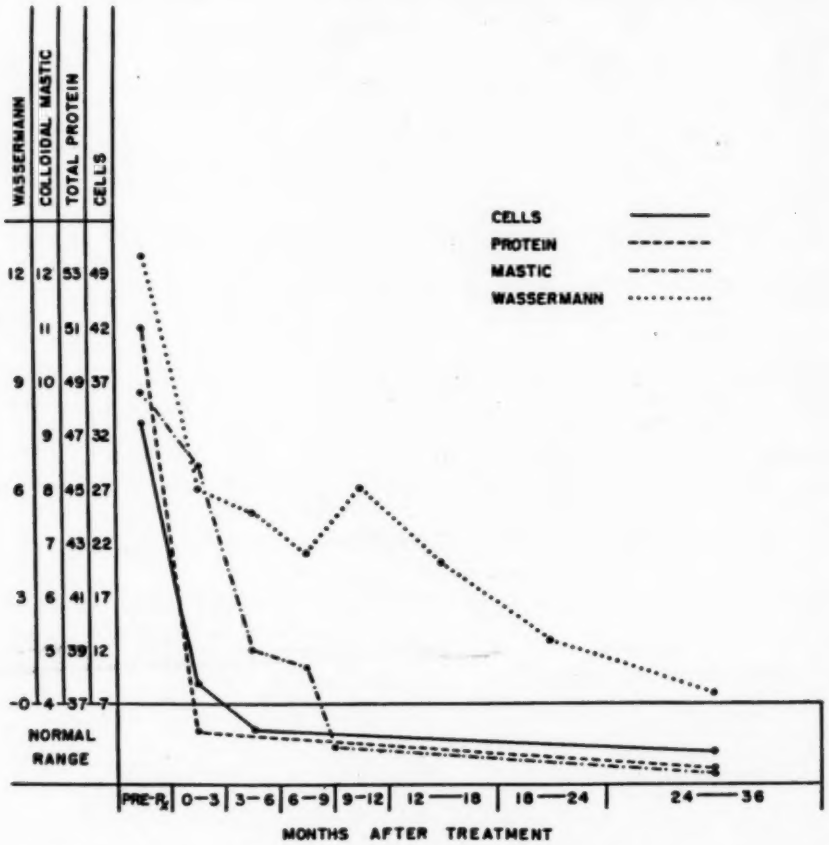

Chart 2.—Effects of therapy with malaria and penicillin on the abnormalities of the cerebrospinal fluid of 21 patients with tabes dorsalis.

(*c*) Effects on the Clinical Manifestations of Tabes Dorsalis: Fourteen (57 per cent) of the 21 patients in group B were adjudged to have shown significant improvement following therapy with malaria and penicillin. The condition of 7 was essentially unchanged. None was worse.

The effects of the combined therapy on the individual symptoms of tabes dorsalis are shown in table 6. It is notable that, as compared

with the results with penicillin alone, a higher proportion of patients showed improvement with respect not only to lightning pains and gastric symptoms, but other symptoms as well.

There were 5 patients, all with symptoms of less than nine months' duration, whose objective physical signs became less strongly abnormal after treatment. In 1 patient the pupillary reaction to light returned; in 2, improved vibratory and/or deep pain sense resulted; in 1, previously absent ankle jerks were elicited, and in another, an area of hyperesthesia to pain and touch stimuli could no longer be demonstrated.

RESULTS OF PENICILLIN THERAPY ALONE AS COMPARED WITH THOSE OF COMBINED PENICILLIN AND MALARIA THERAPY

Although the number of patients treated is small and the period of post-treatment observation far too brief to permit definitive, statistically valid conclusions, it is of interest to compare the results of treatment

TABLE 7.—*Summary of Symptomatic Effect of Penicillin Alone Versus Malaria and Penicillin Therapy on Tabes Dorsalis*

Treatment	Number of Recorded Symptoms	Percentage Showing		
		Significant Improvement	No Significant Change	Worse
Penicillin alone	58	34.4	50.0	15.6
Malaria plus penicillin	52	65.4	32.7	1.9

with penicillin alone and the results obtained with combined malaria and penicillin therapy.

In comparing the two groups of patients, the following factors of selection may be noted: 1. Age and sex distributions were comparable, but the factor of race selection was present, but 1 Negro patient being treated with malaria and penicillin. 2. The duration of symptoms prior to therapy was approximately the same in the two groups. 3. The extent of the pretreatment abnormalities of the cerebrospinal fluid and the ratio of "active" to "inactive" spinal fluids were comparable. 4. No significant difference based on the amount of or response to previously administered antisyphilitic therapy was apparent. 5. Because experimental treatment with malaria and penicillin was started after treatment with penicillin alone, the number of patients in group B is smaller and the duration of post-treatment observation generally shorter than in the corresponding values for group A. All in all, it is believed that the two groups are sufficiently similar to make valid their comparison.

From the standpoint of symptomatic improvement, the results are strikingly better after combined malaria and penicillin therapy than after penicillin therapy alone (table 7). The response in the abnormali-

ties of the cerebrospinal fluid (charts 1 and 2) also is more satisfactory after the combined therapy, although here the difference is less striking.

In rendering the spinal fluid of tabetic patients "inactive," penicillin alone was efficacious, although somewhat less surely and less promptly so than was malaria and penicillin (table 8).

With penicillin therapy alone, the best clinical results were noted in patients in whom the manifestations of tabes were of comparatively recent onset, in whom there were evidences of "activity" in the spinal fluid prior to therapy and in whom the response of the cerebrospinal fluid was promptly favorable.

With combined malaria and penicillin therapy, favorable clinical responses were obtained nearly as often in patients whose symptoms were of long standing as in those whose symptoms were of recent appearance. Moreover, although the majority of the patients whose

TABLE 8.—*"Activity" of the Spinal Fluid Following Therapy with Penicillin Alone and with Malaria Plus Penicillin*

	Pretreatment		0-3 Months		3-6 Months		6-9 Months	
	Total	Number "Active"	Total	Number "Active"	Total	Number "Active"	Total	Number "Active"
Penicillin alone.....	33	20 (60%)	27	7* (26%)	20	3† (15%)	14	2 (14%)
Malaria and penicillin.	21	13 (62%)	10	1† (10%)	8	0	7	0

* In 2 cases "inactivity" of the spinal fluid was subsequently attained without further therapy.

† In 1 case "inactivity" of the spinal fluid was subsequently attained without further therapy.

condition was "significantly improved" had evidence of "activity" in the spinal fluids, there were 2 whose spinal fluids were entirely normal. In all others in whom amelioration of symptoms was noted, there was prompt improvement in the abnormalities of the cerebrospinal fluid.

EFFECTIVENESS OF THERAPY WITH PENICILLIN ALONE AND WITH MALARIA AND PENICILLIN AS COMPARED WITH THAT OF PREVIOUSLY AVAILABLE FORMS OF THERAPY FOR TABES DORSALIS

Prior to the advent of penicillin in syphilotherapy, the treatment of tabes dorsalis entailed the use of trivalent arsenicals and bismuth preparations; pentavalent arsenicals, such as tryparsamide U. S. P. and phenarsone sulfoxylate N. N. R., and malarial therapy, frequently in that order. The efficacy of these schedules of treatment was evaluated in 1938 by the Cooperative Clinical Group.[8]

8. O'Leary, P. A.; Cole, H. N.; Moore, J. E.; Stokes, J. H.; Wile, U. J.; Parran, T.; Vonderlehr, R. A., and Usilton, L. H.: Cooperative Clinical Studies in the Treatment of Syphilis: Tabes Dorsalis, Ven. Dis. Inform. **19**:367 (Nov.) 1938.

TABLE 9.—*Improvement in Tabetic Symptoms Resulting from Therapy with Penicillin Alone: Reports from Four American Clinics*

	Number of Patients	Symptoms or Signs													
		Lightning Pains		Ataxia		Urinary Complaints		Gastric Symptoms		Impotence		Paresthesias		Cranial Nerve Palsies	
		No. Patients	No. Improved	No. Patients	No. Improved	No. Patients	No. Improved	No. Patients	No. Improved	No. Patients	No. Improved	No. Patients	No. Improved	No. Patients	No. Improved
Duke University:															
Callaway and others [9]	26	3	1	10	6	6	5	4	3	1	0	..	..	..	..
University of Pennsylvania:															
Gammon and others [9b]	55	40	33	33	18	18	5	3	2	19	6	..	..	..	..
Cornell University:															
Koteen and others [9c]	41	24	11	12	6	6	2	3	0	6	2	4	4	2	2
Johns Hopkins University:															
Chesney and Reynolds	33	20	9	10	2	11	2	5	3	4	1	7	2	1	1
Totals	155	87	54	65	32	41	14	15	8	30	9	11	6	3	3
			62%		49%		34%		53%		30%		55%		100%

A valid comparison of the results of penicillin therapy with these data necessitates combining the results described by other penicillin investigators with our own. There are in the literature the reports of three clinics [9] in which the effects of penicillin on the various symptoms of tabes are given in detail.[10] The results of these three clinics combined with our own are shown in table 9. We know of no published data on the effects of malaria and penicillin [11] with which to combine our own.

When a comparison is made of the clinical results in treatment of tabes dorsalis with penicillin, combined malaria and penicillin therapy and older forms of therapy (table 10), there is suggestive evidence that penicillin therapy alone is somewhat inferior to therapy of the prepenicillin era, whereas the results with combined malaria and penicillin

TABLE 10.—*Percentages of Patients with Lightning Pains or Ataxia Improved with Various Forms of Therapy*

	Prepenicillin Treatment Schedules *	Penicillin Therapy †	Malaria and Penicillin Therapy ‡
Lightning pains	66	62	87
Ataxia	56	49	64

* Results from the Cooperative Clinical Group.
† Results from Duke University, University of Pennsylvania, Cornell University and Johns Hopkins University.
‡ Results from Johns Hopkins University.

9. (*a*) Callaway, J. L.; Noojin, O.; Flower, A. H., Jr.; Kuhn, B. H., and Riley, K. A.: The Use of Penicillin in the Treatment of Syphilis of the Central Nervous System, Am. J. Syph., Gonor. & Ven. Dis. **30**:110 (March) 1946. (*b*) Gammon, G. D.; Stokes, J. H.; Steiger, H. P.; Steele, W. H.; Beerman, H.; Ingraham, N. R., Jr.; György, P.; Rose, E.; Lentz, J. W.; Ornsteen, A., and Scott, D.: Penicillin Therapy Alone in Neurosyphilis: An Analysis of Clinical Results, Ann. Int. Med. **25**:412 (Sept.) 1946. (*c*) Koteen, H.; Doty, E. J.; Webster, B., and McDermott, W.: Penicillin Therapy in Neurosyphilis, Am. J. Syph., Gonor. & Ven. Dis. **31**:1 (Jan.) 1947.

10. The following articles are also cited: Stokes, J. H., and others: Penicillin in Late Syphilis: An Interim Report, Am. J. Syph., Gonor. & Ven. Dis. **29**:313 (May) 1945. Goldman, D.: Treatment of Neurosyphilis with Penicillin: A Preliminary Report, J. A. M. A. **128**:274 (May 26) 1945. Gammon, G. D., and others: Penicillin in Neurosyphilis: Effect on Blood and Spinal Fluid, ibid. **128**: 653 (June 30) 1945. Rose, A. S., and others: Penicillin Treatment of Neurosyphilis: A Preliminary Report of Seventy Cases Followed from Four to Twelve Months, Am. J. Syph., Gonor. & Ven. Dis. **29**:487 (Sept.) 1945. O'Leary, P. A.; Brunsting, L. A., and Ockuly, O.: Penicillin in the Treatment of Neurosyphilis, J. A. M. A. **130**:698 (March 16) 1946. Stokes, J. H.; Steiger, H. P., and others: Penicillin Alone in Neurosyphilis, ibid. **131**:1 (May 4) 1946.

11. In their reports to the National Research Council, Wile and Curtis (University of Michigan) have stressed the superiority of malaria and penicillin together over penicillin alone in the treatment of various forms of neurosyphilis.

therapy appear superior to both penicillin therapy alone and to previous treatment schedules as defined by the Cooperative Clinical Group.

It should be pointed out, however, that such benefits as were derived from penicillin alone were attained at practically no risk [12] and only slight inconvenience to the patient. Even in expert hands, both metal chemotherapy and malaria therapy entail greater inconvenience and a mortality rate which, although small, is not negligible. For this reason, and because tabes dorsalis involves no immediate threat to life, Moore [13] suggested that penicillin be employed first, the use of malaria (with more penicillin?) being reserved for those patients who after an observation period of six months fail to improve significantly.

SUMMARY AND CONCLUSIONS

Fifty-four patients with tabes dorsalis were treated with penicillin. Thirty-three of these received penicillin alone; 21, penicillin concurrently with therapeutic malaria.

With penicillin therapy alone, the preliminary results from this clinic indicate that the symptoms of 27 per cent of patients were significantly improved. The effects on the cerebrospinal fluid were favorable in a high percentage of patients, although "inactivity" was not invariably attained. Febrile Herxheimer reactions occurred only in patients with spinal fluids which gave strongly positive reactions for syphilis and were far less frequent than among patients with dementia paralytica.

With malaria and penicillin therapy, 57 per cent of our patients were adjudged to have shown significant symptomatic improvement. The effects on the cerebrospinal fluid were uniformly favorable.

A comparison of the results with penicillin alone and those with malaria and penicillin indicates clearcut superiority for the combined therapy.

Penicillin treatment alone appears superior to therapy of the prepenicillin era (as defined by the Cooperative Clinical Group) only in respect to its convenience and relative lack of toxicity.

Concurrently administered penicillin and therapeutic malaria is the most effective form of therapy for tabes dorsalis presently available.

804 Medical Arts Building.

12. In the series reported by Koteen and his co-workers, there were 2 cases of tabes dorsalis with death within two weeks of penicillin therapy. In both, death was preceded by high fever (42 C. [107.6 F.]), with progressive stupor and terminal coma.

13. Moore, J. E.: Penicillin in Syphilis, Springfield, Ill., Charles C Thomas, Publisher, 1947.

SUBDURAL HEMATOMA

An Experimental Study

ROBERT COHN, M.D.
BETHESDA, MD.

DURING the past years a large number of persons with recent and old head injuries have been referred to the electroencephalographic department for assistance in the differential diagnosis of subdural hematoma from other forms of intracranial lesions. At first the criterion of Jasper,[1] namely, decreased amplitude of electric activity over the region of hemorrhage, was relied on. However, it was soon recognized that verified cases of subdural hemorrhage often gave rise to generalized high voltage, slow waves with a focus of maximum abnormality over the region of the extravasation of blood. Moreover, occasional records were obtained in which no definite focal or general abnormality was demonstrated. It became imperative, therefore, to attempt to establish the basis for these observed electroencephalographic findings in man. For this purpose, animal experiments were designed in which the controlled introduction of the animal's own blood into the intracranial cavity simulated the condition of spontaneous subdural hemorrhage in man.

METHOD

Thirty-two cats of average size were used. The electrodes (fig. 1) were fixed in the calvaria twenty-four to forty-eight hours prior to the production of the intracranial hemorrhage. They were placed in homologous frontal, parietal and occipital regions over the cerebral hemispheres. With pentobarbital anesthesia and surgical asepsis, a circular cutter removed small areas of scalp and underlying muscle. With a drill guard to prevent laceration of the brain, holes were drilled of sufficient size for insertion of an 8-32 thread tap. All drilled and tapped holes were carefully washed with sterile saline solution before securing the lucite-enclosed electrodes in place. At the time of placement of the electrodes another tap was made in the skull to insert the inlet adapter for the introduction of whole blood

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This article has been released for publication by the Division of Publications of the Bureau of Medicine and Surgery of the United States Navy. The opinions and views set forth in this article are those of the writer and are not to be considered as reflecting the policies of the Navy Department.

1. Jasper, H. H.; Kershman, J., and Elvidge, A.: Electroencephalographic Studies of Injury to the Head, Arch. Neurol. & Psychiat. **44**:328-350 (Aug.) 1940.

into the subdural space. This hole was made in the right or the left frontal or parietal region. Before the inlet adapter was fixed in place, the dura was incised with a flat twist drill. The shank of the adapter was sealed off with a threaded lucite cap. While the animal was still under anesthesia, approximately 10 cc. of cardiac blood was withdrawn. The blood was refrigerated and kept from coagulating with sodium citrate.

When the cat was again alert (one or two days after the implantation of electrodes), 2 to 4 cc. of the citrated blood was introduced subdurally. It was found that an excess of 4 cc. of blood invariably resulted in acute respiratory death of the animal.

Control electroencephalograms were made before the blood was introduced, during the production of the "hemorrhage" and intermittently for three or four hours afterward (acute experiments). In chronic experiments, after control runs, recordings were made for several hours immediately after the injection of blood and thereafter one to three times daily up to fourteen days.

In most of the experiments both bipolar and monopolar derivations were used. Often the bipolar derivations gave a more distinct and easily observed localization of the disturbed cerebral function than the monopolar derivations. All recordings were made with a Grass six channel electroencephalograph.

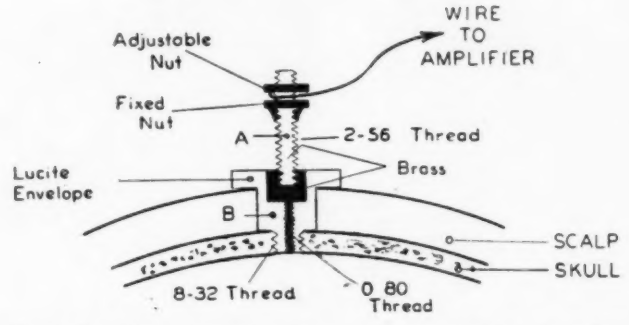

Fig. 1.—The indwelling electrode. Part *A* is detachable. It is screwed into *B* during recording.

The animals which survived the effects of the "hemorrhage" were killed by intracardiac injection of pentobarbital. The brains were immediately perfused with saline solution, followed with dilute solution of formaldehyde U.S.P. (1:20). After removal of the brain the site and extent of the "hemorrhage" (fig. 2 *A*, *B*, *C* and *D*) were determined. In all experiments in which convulsions occurred and in all chronic experiments, histologic studies were carried out. Appropriate stains for cells and myelin sheaths were used in the histologic study.

RESULTS

Subdural introduction of blood in the frontal region almost immediately gave rise to notable increases in the respiratory and cardiac rates. This reaction was most intense when the injected blood covered the lateral and inferior frontal surfaces of the brain. Together with these responses, most animals became obviously unaware of their surroundings. They did not respond to strong tactile or auditory stimuli. This phase usually persisted for several hours. Within one-

half hour after the injection, hemiplegias (in extension) were observed on the side opposite the lesion. In many cats sudden ragelike reactions occurred within the first three hours. The rapid respiratory rate usually subsided spontaneously within six to eight hours. Variation in pupillary size during the acute phase was not constant, but dilatation on the

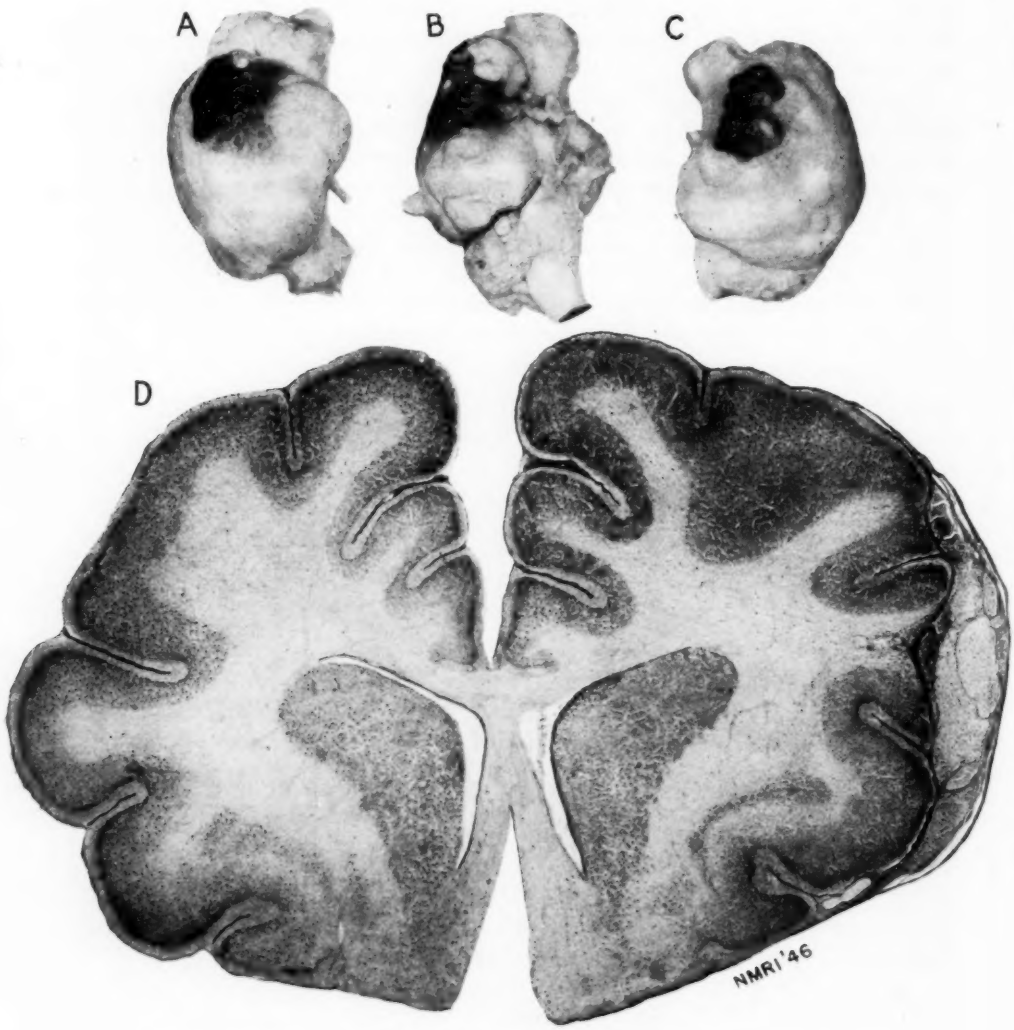

Fig. 2.—Gross appearance of experimental subdural hematoma in cats.

side of the hematoma was most usual, although bilateral dilatation was prominent. The whisker reflex was nearly always absent contralateral to the "hemorrhage." The neurologic signs, including stepping and hopping reactions, gradually decreased in intensity but did not completely disappear even in the longest experiments. The observed clinical display,

particularly that referable to the autonomic nervous system, was entirely similar to the behavior described by Kennard [2] in cats with acute bilateral decortication.

Subdural injection of blood in the parietal region gave rise to immediate transient bilateral motor deficits (usually flexion paraplegias of the forelimbs) and in each animal produced bilateral internal ophthalmoplegia and apparent blindness. As the preparations became chronic, it was not always possible to determine whether there was complete bilateral amaurosis or only homonymous field defects. The animals would walk into objects or would walk in a circle. When placed in a relatively high-walled box, these animals would stand on an edge and make strong attempts to touch the floor with the forepaws. At no time would they leave their precarious perch unless they could feel a solid object.

Four cats in this series showed generalized convulsive seizures. In 3 cats the seizures occurred within the first thirty minutes after the instillation of subdural blood. The other animal had onset of seizures three days after the "hemorrhage" and died in status epilepticus.

Immediately after the administration of blood, no variations in electric potentials could be elicited (fig. 3 *B*). Usually within one to three minutes the "no" potential interval was followed by high voltage, slow waves of random frequency over the side opposite the "hemorrhage." The side of the "hemorrhage" remained essentially isoelectric (fig. 3 *C*). Often, within five minutes after the introduction of blood the electric output of the entire brain was of high voltage and low frequency. The waves were slower and smoother (less superimposed oscillations) over the site of the lesion (fig. 3 *D*). Approximately ten minutes after the introduction of blood into the subdural space, bilateral low voltage, fast waves were observed, and volleys of asymmetrically peaked waves (spikes) were occasionally seen over the side of the lesion (fig. 3 *E*). Subsequent to this, focal 1½ to 2 per second waves were recognized over the insulted cerebral hemisphere (fig. 3 *F*). As time progressed, the tracings from the side of the hematoma continued to show low voltage, random potentials of medium frequency; the potentials from the contralateral hemisphere gradually became lower in voltage and higher in frequency, until approximately one week after the "hemorrhage" the control type of pattern was regained (fig. 3 *G*, *H*, *I*, and *J*).

Figure 4 shows a sample electroencephalographic record for a cat in which a right frontoparietal (fig. 2 *B*) subdural "hemorrhage" was established. It may be observed that the majority of the slow waves occurred in the right occipital region. Only occasionally was low frequency activity recognized in the parietal derivation. As chronicity

2. Kennard, M. A.: Focal Autonomic Representation in the Cortex and Its Relation to Sham Rage, J. Neuropath. & Exper. Neurol. **4**:295-304, 1945.

progressed, the amplitude of electric output was increased to a greater degree over the right frontal region than over the left; together with this moderate asymmetry in amplitude, the frequency ranged about 12 per second on the right, with random, faster activity on the uninjured side.

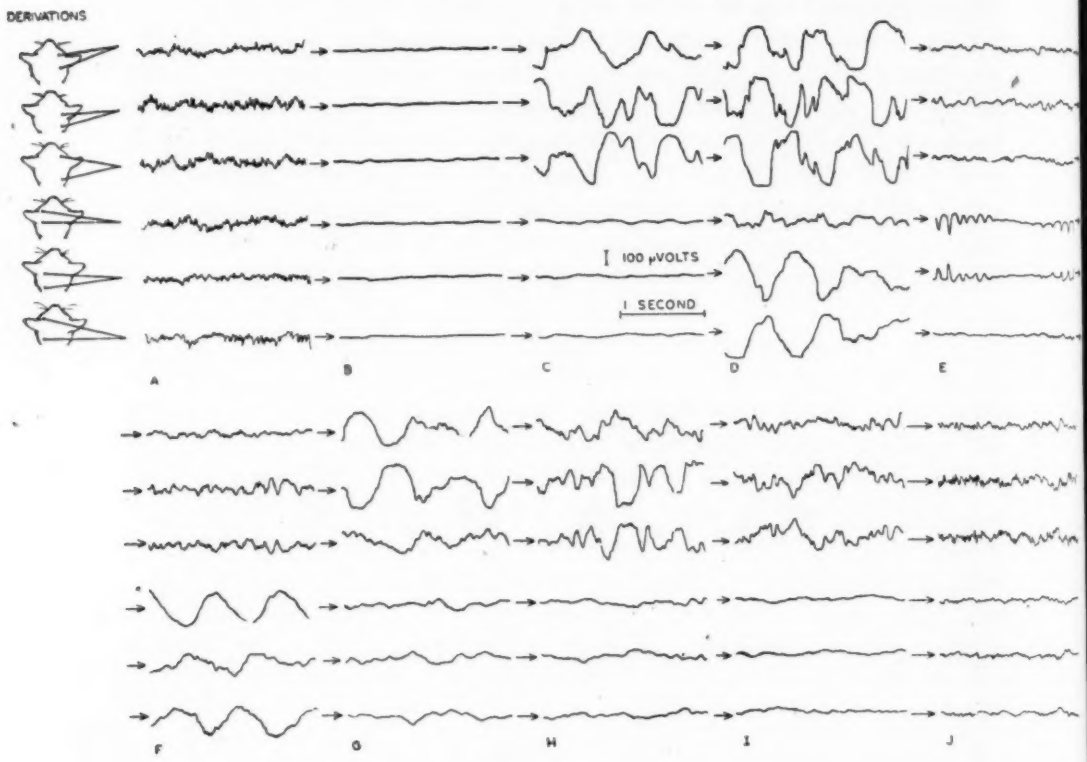

Fig. 3.—(*A*) control tracing. (*B*) Sixty seconds after the instillation of blood in the subdural space no electric potentials are observed in any derivation. (The "hemorrhage" was introduced in the left frontal region.) (*C*) Approximately sixty seconds later the "no" potential activity persists over the site of "hemorrhage." The contralateral cerebral hemisphere shows random high voltage, low frequency oscillations. (*D*) Five minutes after the introduction of blood the insulted cerebral hemisphere shows low frequency waves, particularly in the occipital region. The cortex of the opposite side also gives rise to slow waves, but activity of higher frequency is superimposed on the basic oscillation. (The phase difference of lines 5 and 6 is a phenomenon of electrode combination and is not of primary importance.) (*E*) Eleven minutes after the production of the subdural hematoma the uninjured cerebral hemisphere shows a pattern of very low voltage and random frequency. On the side of the "hemorrhage" an asymmetric, spikelike discharge is generated over the frontal region. This type of wave was occasionally associated with generalized involuntary motor discharges. (*F*) About nineteen minutes after *E* was recorded, rhythmic high voltage, slow waves dominate the left frontal potential output. The "undamaged" cerebral hemisphere shows a beginning return to the control type pattern. (*G*, *H* and *I*) Samples of record taken approximately forty, one hundred and thirty and three hundred and seventy minutes after the instillation of subdural blood show that decreased electric output over the site of "hemorrhage" persists. The contralateral cerebral hemisphere gives rise to prominent slow activity, which progressively approaches the control type pattern. (*J*) Seven days after "hemorrhage" a low voltage output is manifest over the insulted cerebral hemisphere. The contralateral hemisphere shows a return to the control electroencephalographic pattern.

Microscopic studies showed no uniform picture in the animals which had convulsions within thirty minutes after the subdural hematoma. Two of these animals showed entirely normal parenchyma in the region subjacent to the hematoma. One had slight perivascular infiltration with polymorphonuclear cells and engorgement of the capillary beds in the cortex underlying the hematoma. In the white matter of the affected side many gitter cells as well as defense reaction cells were recognized. The defense reaction extended into the molecular layer of the cortex. The entire damaged left cerebral hemisphere was observed to be swollen when compared with the right hemisphere.

In the chronic experiments there was a correlation of the first order between pathologic changes and the observed potential output. The cats with extensive focal loss of cells underlying the hematoma had a persistent decrease of electric activity over the site of the hematoma.

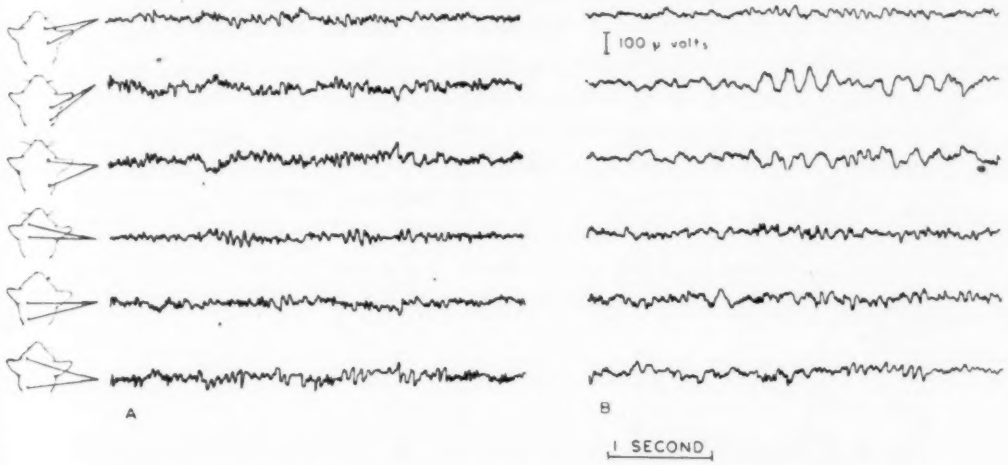

Fig. 4.—(*A*) Control pattern. (*B*) Ten days after the "hemorrhage" (right frontoparietal region) the insulted cerebral hemisphere gives rise to generalized volleys of slow waves, more often from the right occipital derivation. Only occasionally were frontoparietal (right) slow waves observed.

The cats that had moderate degree of cortical cell death and an active proliferative defense reaction gave rise to transient, but prominent, high voltage, slow waves over the damaged, as well as the undamaged, cerebral hemisphere, together with a decrease in detail of the potentials (lack of high frequency activity) over the injured side. In animals in which a small number of cortical cells were irreversibly damaged, after the initial electric changes (fig. 3), an essentially control type of pattern usually developed within a week after the instillation of the blood into the subdural space.

COMMENT

From the results it is clear that the initial response of the cortical neurons to the "hemorrhage" was shock. This phenomenon was

entirely similar to the effects observed after any traumatic insult to the brain, such as a blow to the head.[3] This phase of shock was at least transient in all experiments with subdural hematoma. Jasper [1] expressed the belief that the shock reaction was the chief characteristic of subdural hematoma in man. He attributed the decreased electric activity observed to the interposition of a short circuiting mass of blood between the brain and the tissue elements, through which the biopotentials are derived.

In the present communication, the interval of decreased electric activity over the insulted side of the brain appeared to depend on whether the brain cells were reversibly or irreversibly damaged. When damage was irreversible, the findings of Jasper held true. However, when no large mass of cells was destroyed, after a variable interval of extremely low voltage (or no) potential output, low frequency activity over, or adjacent to, the site of the "hemorrhage" was the rule. This phenomenon was even more definite in the human cases studied.

Hence, as a result of the correlation of the gross electric and the histologic changes, it is believed that the sequence of events in the cortical neurons following subdural hemorrhage is (1) shock state (no potential output or a greatly reduced amplitude of electric output); (2) a "sick" brain (slow waves), and (3) recovery, as evidenced by the return of the pattern to "normal," or death of a substantial number of neurons, manifesting itself in continued lower voltage of, and persistent lack of detail in, the potential output over the involved portion of the brain.

It is clear from both the electrical behavior and the histopathologic observations that subdural hematoma is not a precise clinical entity. The clinical signs and symptoms, as well as the electric activity observed, depend on the location, the volume of blood and the intensity of the resultant damage to nerve cells produced by the subdural hemorrhage.

CONCLUSIONS

With acute experimental subdural hematoma there is decreased electric activity over the site of "hemorrhage."

In subdural "hemorrhages" of one week or more in duration (*a*) the focal decreased electric activity of the acute phase may persist in varying degrees; (*b*) there may be generalized slow waves with a focus of maximum abnormality over the "hemorrhage" or adjacent areas, or (*c*) an essentially normal electroencephalographic pattern may be observed.

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3. Williams, D., and Denny-Brown, D.: Cerebral Electrical Changes in Experimental Concussion, Brain **64**:223-228, 1941. Shelden, C. H.; Pudenz, R. H., and Cohn, R.: Experimental Head Injury, unpublished data.

It is postulated that the sequence of events occurring in the cortical neurons following the local implantation of blood into the subdural space is as follows: (*a*) shock state as evidenced by no electric output or a greatly reduced amplitude of potential; (*b*) a condition of sick brain cells, as indicated by slow wave activity, and (*c*) death of a substantial number of neurons, with persistent lack of detail in the potential output over the involved portion of the brain, or (*d*) recovery, with return of the pattern to its control type.

There are no unique electroencephalographic findings associated with the experimental subdural hematoma. This results from the fact that the electric output of the brain is a function of the areal exent and the degree of injury to the cortical neurons produced by the overlaying blood mass.

The instillation of blood into the subdural space of cats generally gives rise to clinical phenomena that are similar to those observed after acute decortication.

DISTRIBUTION OF LESIONS OF THE BRAIN STEM IN POLIOMYELITIS

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J. C. McCARTER, M.D.

AND

H. W. MAGOUN, Ph.D.

CHICAGO

THE BEST established feature of the neuropathology of poliomyelitis is the destruction of anterior horn cells of the spinal cord, this being generally recognized as the injury responsible for the enduring paralysis and atrophy of muscles of the trunk and extremities which may occur in this disease. Similarly, when present, paralysis of the cranial musculature or that of the oropharynx has been attributed to involvement of the analogous motor nuclei of the cranial nerves in the brain stem. As a consequence, and in spite of much evidence opposing such a view (Howe and Bodian[1]), there appears to be at least the popular impression that the only symptomatically important injury to the nervous system associated with poliomyelitis is the decrease in motor neuron collections.

This concept was recently challenged by Kabat and Knapp,[2] who called attention to the frequent involvement of interneurons in the gray matter of the spinal cord, suggesting that this was the factor responsible for the "muscle spasm" of this disease. Our interest in the distribution of poliomyelitic lesions at central levels above the cord was aroused by a demonstration by Dr. A. Weil of conspicuous injury to the bulbar reticular formation in a fatal case, for this raised two questions: Is injury to supranuclear neural collections in the brain stem of frequent occurrence in poliomyelitis? If so, what role may it play in the course of the illness?

From the Departments of Anatomy and Pathology, Northwestern University Medical School, and the Evanston Hospital Association.

The study was aided by a grant to the Northwestern University Medical School from the National Foundation for Infantile Paralysis, Inc.

1. Howe, H. A., and Bodian, D.: Neural Mechanisms in Poliomyelitis, New York, The Commonwealth Fund, 1942.

2. Kabat, H., and Knapp, M. E.: The Mechanism of Muscle Spasm in Poliomyelitis, J. Pediat. **24**:123-137, 1944.

While the second of these questions can at present be discussed only inferentially, the results detailed in this paper contribute evidence for a definitely affirmative answer to the first. Examination of serial sections of the brain stems in 7 fatal cases of poliomyelitis, the deaths occurring in the summers of 1943, 1944 and 1945, revealed in each instance a columnar range of supranuclear lesions in the reticular formation and tegmentum forming the central core of the brain stem. Seven cases might, in themselves, be too small a group on which to base a general conclusion, but a sufficient number of additional references to this characteristic injury have been found in the literature to suggest that it is a common, if not an invariable, feature at least of that proportion of cases of poliomyelitis progressing to an acute fatality.

METHODS

After the entire central nervous system was removed at autopsy and fixed in a dilute solution of formaldehyde U.S.P., blocks including representative segments of the spinal cord, the entire brain stem, the central nuclei of the cerebellum and portions of the cerebral cortex were subsequently embedded in pyroxylin and sectioned serially at a thickness of 50 or 75 microns. Every twelfth section was mounted after being stained with the buffered thionine method of Windle, Rhines and Rankin.[3] Intervening, thinner sections stained with hematoxylin and eosin were also available.[4]

In addition to microscopic examination, to which all the material was subjected, enlarged projection drawings of sections of the brain stem were prepared on which the distribution of lesions was indicated. In each case these figures were employed in transposing the range of lesions to a reconstruction of the midsagittal plane of the brain stem, and in 1 case transference of these figures to sheets of "pyralin" made possible the preparation of a three dimensional, transparent model of the brain stem with the distribution of lesions indicated within it.

RESULTS

Nature of Lesions.—The neuronal changes induced by poliomyelitis virus proceeded from chromatolytic alterations through ghost cell formation to disappearance of the neuron. The inflammatory reaction evoked by this necrotic process was only initially one of polymorphonuclear leukocytes; at the stage to which the disease in the present cases had progressed, it consisted of dense focal and more diffuse parenchymal accumulations and perivascular cuffs of mononuclear cell types, evidently contributed to by monocytes and lymphocytes from the blood stream and by microglia from the neural parenchyma. Diffuse inflammation and perivascular cuffing were rather generally distributed through an affected region, the cuffs in particular often extending beyond it into otherwise normal areas. Dense inflammatory foci in the parenchyma,

3. Windle, W. F.; Rhines, R., and Rankin, J.: A Nissl Method Using Buffered Solutions of Thionin, Stain Technol. **18**:77-86, 1943.

4. Miss Doris Dieskow prepared most of this large collection of slides.

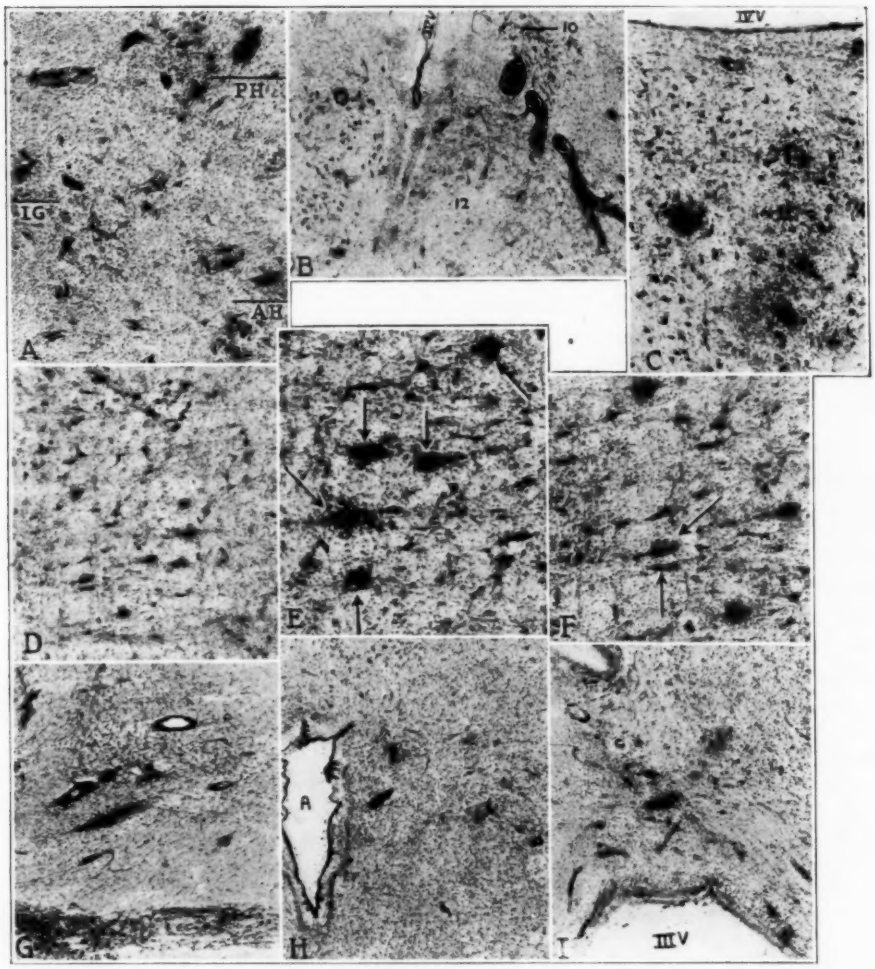

Fig. 1.—Photomicrographs of lesions of poliomyelitis in the spinal cord (*A*), the hypoglossal nucleus (*B*), the vestibular nucleus (*C*), the reticular formation of the medulla (*D* to *F*), the pontile tegmentum (*G*), the periaqueductal gray matter (*H*) and the wall of the third ventricle (*I*) of the human brain in acutely fatal cases.

Abbreviations used in this figure, and/or in the accompanying figures, are as follows: *A*, aqueduct; *AC*, anterior commissure; *AH*, anterior horn; *BC*, brachium conjunctivum; *BP*, basis pedunculi; *CP*, cerebellar peduncle; *HY*, hypothalamus; *I*, infundibulum; *IC*, internal capsule; *IG*, intermediate gray matter; *M*, mamillary body; *MG*, medial geniculate body; *ML*, medial lemniscus; *MLF*, medial longitudinal fasciculus; *MP*, middle peduncle, or brachium pontis; *O*, inferior olive; *OT*, optic tract; *P*, pons; *PC*, posterior commissure; *PH*, posterior horn; *PN*, nuclei of the posterior column; *PY*, pyramid; *R*, restiform body; *RN*, red nucleus; *S*, subthalamic nucleus; *SC*, superior colliculus; *SN*, substantia nigra; *SV*, spinal trigeminal tract; *TH*, thalamus; *TS*, tractus solitarius; *III V*, third ventricle; *IV V*, fourth ventricle; *3*, oculomotor nucleus; *5*, motor nucleus of the trigeminal nerve; *10*, motor nucleus of the vagus nerve; *12*, hypoglossal nucleus.

on the other hand, were more immediately confined to regions of neuronal destruction, and sharply delimited concentrations of inflammatory cells frequently marked the former positions of large neurons.

Representative examples of this focal parenchymal inflammation, and the neuronal destruction with which it was associated, are seen at various levels of the spinal cord and brain stem in figure 1. In the cord, injury was by no means confined to the anterior horns but frequently involved the intermediate, and sometimes the posterior, gray matter (fig. 1 *A*). In the brain stem, an example of involvement of the motor nuclei of cranial nerves, with adjacent perivascular cuffing, is seen in figure 1 *B*, in which many neurons of the right hypoglossal nucleus are ghostlike, while the adjacent dorsal nucleus of the vagus nerve and the left hypo-

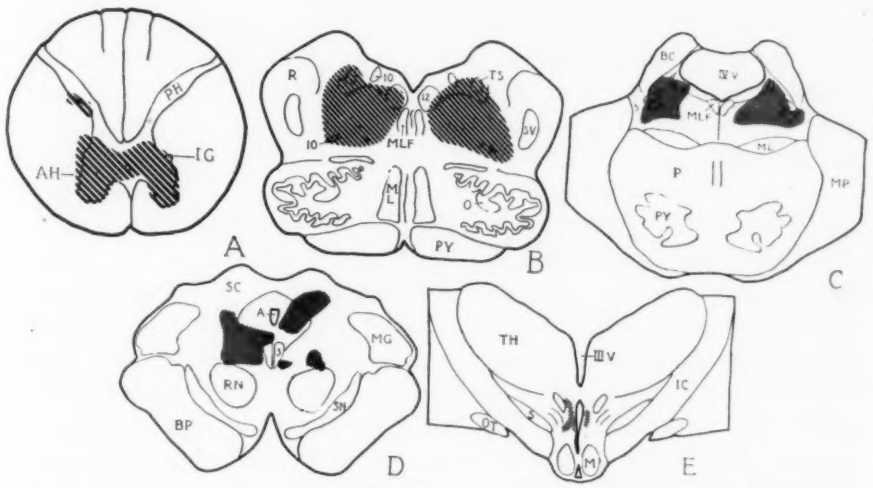

Fig. 2.—Transverse sections through the spinal cord (*A*) and brain stem (*B* to *E*) of a fatal case of poliomyelitis, with the distribution of lesions indicated by cross lining.

glossal nucleus are little affected. Inflammatory foci in the vestibular nuclei, the most frequently involved of the cranial sensory cell groups, are seen in figure 1 *C*.

The involvement of the bulbar reticular formation (fig. 1 *D* to *F*) usually equaled in severity that of the gray matter of the spinal cord. The view taken with the low power lens in figure 1 *D* illustrates the extensive range of inflammatory foci in the bulbar reticular area, and the diffuse inflammation present may be gaged by comparing the background here (fig. 1 *D*) with that in other regions of the brain stem (fig. 1 *G* to *I*). The more magnified field, in figure 1 *E*, shows instances of phagocytosis of necrotic reticular neurons (arrows); other such instances are seen in figure 1 *F*, in which two intact, but severely chromatolyzed, neurons (arrows) can still be distinguished.

At more rostral levels, a focal inflammatory process in the tegmental portion of the pons is shown in figure 1 *G*, in which is also indicated the absence of involvement of the base of the pons appearing in the lower part of the figure. Smaller foci in the periaqueductal gray matter of the midbrain (fig. 1 *H*) and the periventricular gray matter of the diencephalon (fig. 1 *I*) illustrate the characteristically diminishing extent and severity of lesions at these rostral levels of the brain stem.

Distribution of Lesions.—In figure 2, cross lining is employed to indicate the range of inflammatory foci, exemplified by figure 1, at transverse levels through the brain stem in 1 case (*44-114*, fig. 3). At levels rostrad to the anterior horns and intermediate gray matter of the spinal cord (fig. 2 *A*), these lesions were distributed through much of the extent of the reticular formation of the medulla oblongata (fig. 2 *B*) and, in an irregular fashion, invaded the cranial nuclei bordering the reticular area dorsally and laterally. At the level of the pons, the lesions were distributed in the pontile tegmentum (fig. 2 *C*), with predilection for its more lateral parts, where the trigeminal nuclei were affected. The basilar part of the pons (fig. 2 *C*), like the inferior olive, behind (fig. 2 *B*), and the cerebral peduncle, ahead (fig. 2 *D*), was free of injury. From the pons, lesions passed forward into the tegmentum of the midbrain and into the periaqueductal gray matter (fig. 2 *D*) and from the latter region into the periventricular portions of the subthalamus and hypothalamus of the diencephalon (fig. 2 *E*). The lesions in this rostral part of the brain stem became somewhat discontinuous, and in general (fig. 1 *H* and *I*) their severity was less than at more caudal levels.

A clearer conception of the continuity of the poliomyelitic injury in the cord with that distributed through the brain stem in each case studied was gained by projecting the range of inflammatory foci at each successive level upon a reconstruction of the midsagittal plane of the neuraxis. Such reconstructions of the brain stems of all 7 cases are presented in figure 3. Lesions were seen not only in the spinal gray matter but extending forward through the central core of the brain stem, in each instance with injury to the bulbar reticular formation, the pontile tegmentum and the central part of the midbrain, and usually to the basal portion of the diencephalon as well. Structures of the brain stem lying ventral to this columnar zone—the inferior olive, the basilar part of the pons and the cerebral peduncle—were unaffected. Structures situated dorsal to it were involved with decreasing frequency rostrally: The central cerebellar nuclei overlying the lower part of the brain stem were injured in 6 cases; the tectum roofing the midbrain, in 4 cases, and less severely, and the thalamus of the diencephalon, in but 1 case. Parenchymal inflammatory foci and cuffing were present in varying parts of the precentral gyrus of the cerebral cortex in 4 cases, being minimal in 3 and severe in the fourth.

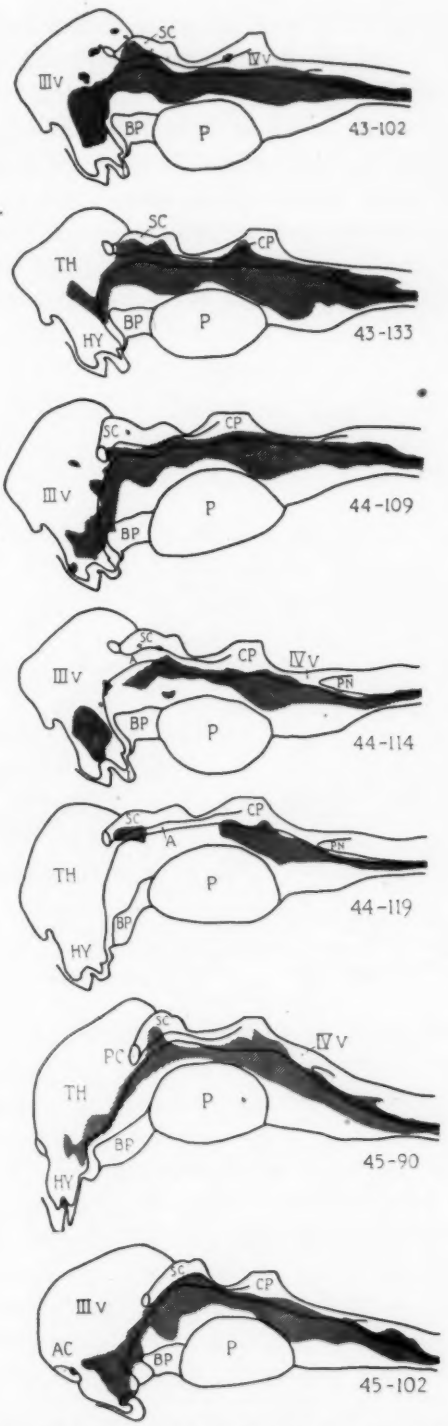

Fig. 3.—Reconstructions of the midsagittal plane of the brain stems in 7 fatal cases of poliomyelitis on which the distribution of lesions has been projected with cross lining.

A three dimensional view of this cranial distribution of poliomyelitic injury in 1 case (*44-114*) was gained by preparing a transparent model of the brain stem with the extent of the injured area indicated within it. A drawing of this model is reproduced in figure 4. Within the phantom outlines of the brain stem, the affected area is seen to extend forward from the gray matter of the spinal cord through the reticular formation of the bulb, the pontile tegmentum and the central portion of the midbrain, to terminate rostrally in the basal portion of the diencephalon in the characteristic manner observed in each of the cases of the present series.

COMMENT

In each of these acutely fatal cases of poliomyelitis, injury, present through extensive anteroposterior distances of the spinal cord, was by

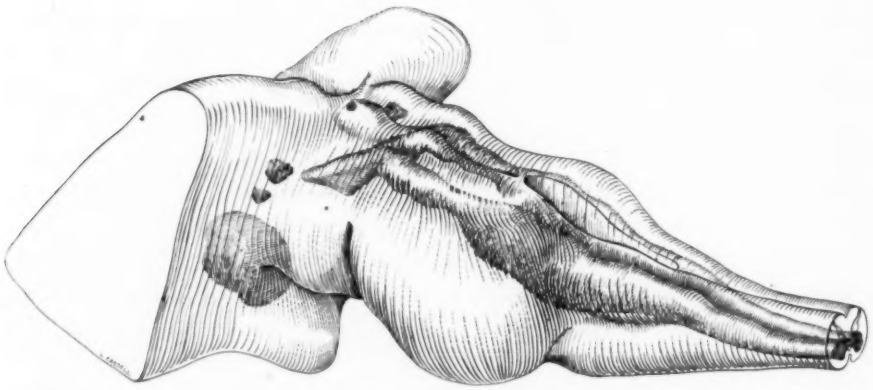

Fig. 4.—Phantom outline of the brain stem of a fatal case of poliomyelitis, with the distribution of lesions indicated within it (drawn from a transparent model by Miss Lucille Cassell).

no means confined to spinal levels but extended forward also through the brain stem practically its entire length. The pattern of distribution of this injury within the brain stem was similar, furthermore, from one case to the next, for the central core, in particular the reticular formation and the tegmentum, was the region characteristically affected.

Though this injury to the brain stem has not, in our opinion, previously received the recognition its characteristic presence merits, it has seemingly been observed, as Wickman [5] remarked, "in every fatal case of poliomyelitis in which it has been sought"; and, Wickman added, "the most intense changes in the bulb appear often apart from the nerve nuclei, in the substantia reticularis tegmenti." Lesions in this region

5. Wickman, I.: Acute Poliomyelitis (Heine-Medin's Disease), Nervous and Mental Disease Monograph 16, New York, Nervous and Mental Disease Publishing Company, 1913.

were reported, illustrated and diagramed in the papers of Kino,[6] Finley[7] and Guizetti,[8] the last author commenting, "Only *one* area [in the brain stem] is always severely altered, and that is the so-called substantia reticularis." Howe and Bodian[1] assigned a prominent role to connections of the reticular formation in discussing the principal routes of dissemination of poliomyelitis virus in the central nervous system. The recent papers of Faber and Silverberg[9] and Luhan[10] emphasized both the involvement of the brain stem and the severity of injury to the reticular formation in fatal cases of poliomyelitis. In 10 fatal cases of poliomyelitis occurring in 1946 in the Evanston Hospital, in which less detailed microscopic examination of the central nervous system was made, severe involvement of the brain stem was observed in 9, the distribution of the lesions conforming to the pattern described here. In 1 case of extremely severe damage to the spinal cord, lesions of the brain stem were minimal.

When the functional significance of the reticular formation and the tegmentum of the brain stem are considered, it would appear that their involvement in poliomyelitis may be of importance in accounting for the fatal course in those cases in which it has been observed. In each of the present cases there was terminal paralysis of respiration, attributable in part at least to injury to the respiratory center, which is located in this portion of the brain stem (Pitts[11]). Each patient was placed in a respirator, however, and oxygen was administered, so that it is not possible to ascribe the fatal outcome to paralysis of respiration alone.

The experimental studies of Wang and Ranson[12] and Alexander[13] indicated that the reticular formation of the lower portion of the brain stem contains mechanisms essential to the normal regulation of vasomotor activity, injury to which may have contributed to death in these cases. The pulmonary edema encountered at autopsy in these cases may have

6. Kino, F.: Die Poliomyelitis des Hirnstammes (Zur Lehre von der Pathoklise), Ztschr. f. d. ges. Neurol. u. Psychiat. **113**:332-347, 1928.

7. Finley, K. H.: The Neuro-Anatomy in Respiratory Failure: Report of Two Cases, Arch. Neurol. & Psychiat. **26**:754-783 (Oct.) 1931.

8. Guizetti, H. U.: Betrachtungen zur Poliomyelitis des Hirnstammes, Deutsche Ztschr. f. Nervenh. **131**:29-42, 1933.

9. Faber, H. K., and Silverberg, R. J.: A Neuropathological Study of Acute Human Poliomyelitis with Special Reference to Initial Lesions and to Various Potential Portals of Entry, J. Exper. Med. **83**:329-354, 1946.

10. Luhan, J. A.: Epidemic Poliomyelitis: Some Pathologic Observations on Human Material, Arch. Path. **42**:245-260 (Sept.) 1946.

11. Pitts, R. F.: Organization of the Respiratory Center, Physiol. Rev. **26**: 609-630, 1946.

12. Wang, S. C., and Ranson, S. W.: Autonomic Responses to Electrical Stimulation of the Lower Brain Stem, J. Comp. Neurol. **71**:437-455, 1939.

13. Alexander, R. S.: Tonic and Reflex Functions of Medullary Sympathetic Cardiovascular Centers, J. Neurophysiol. **9**:205-217, 1946.

had its basis in disturbance of vasomotor regulation; a neurogenic factor in pulmonary edema associated with other conditions has been suggested.[14]

Incoordination or paralysis of the pharyngeal and laryngeal musculature, exemplified by failure of the swallowing reflex, doubtless contributed to the patient's difficulties by preventing the normal excretion of mucus from the pulmonary passages into the alimentary tract, and by permitting aspiration from the oral cavity. It is by no means certain that this deficit resulted solely from direct injury to motor nuclei of the lower cranial nerves, as has been suggested, inasmuch as a mechanism for integrated swallowing has been identified by Rhines [15] in the nucleus solitarius.

The demonstration of severe injury to vital neural mechanisms in the lower portion of the brain stem in fatal cases of poliomyelitis should call attention to the need of basic investigation of these mechanisms, knowledge of which might be expected ultimately to contribute to the clinical management of patients with this disease. Advances in knowledge of extrapyramidal components of the lower portion of the brain stem suggest that injury to this region may be a factor contributing to motor symptoms of poliomyelitis inexplicable in terms of destruction of the anterior horn cells.

A bulboreticular mechanism exerting an inhibitory influence on motor activity (Magoun and Rhines [16]) has recently been found to comprise the extrapyramidal, brain stem relay through which both the suppressor area, 4-s, of the cerebral cortex (McCulloch, Graf and Magoun; [17] McCulloch, Ward and Magoun [18]) and the suppressor portion of the portion of the cerebellum (Snider, McCulloch and Magoun [19]) influence the motor outflows of the spinal cord to prevent hyperreflexia and hypertonicity. Injury to this bulbar inhibitory mechanism was correlated by Bodian [20] with the appearance of generalized spasticity in experimental poliomyelitis in monkeys. Similarly distributed injury to the brain stem was demonstrated in the cases forming the subject of the present report, but the neuropathologic basis for comparable motor symptoms of human

14. Schlesinger, B.: Neurogenic Pulmonary Edema, Due to Puncture Wound of the Medulla Oblongata, J. Nerv. & Ment. Dis. **102**:247-255, 1945.

15. Rhines, R.: Bulbar Structures Involved in Swallowing, Anat. Rec. **97**:87, 1947.

16. Magoun, H. W., and Rhines, R.: An Inhibitory Mechanism in the Bulbar Reticular Formation, J. Neurophysiol. **9**:165-171, 1946.

17. McCulloch, W. S.; Graf, C., and Magoun, H. W.: A Cortico-Bulbo-Reticular Pathway from Area 4-s, J. Neurophysiol. **9**:127-132, 1946.

18. McCulloch, W. S.; Ward, A. A., Jr., and Magoun, H. W.: One Component of the Extrapyramidal System, Tr. Am. Neurol. A. **71**:93-96, 1946.

19. Snider, R. S.; McCulloch, W. S., and Magoun, H. W.: A Cerebello-Bulbo-Reticular Suppressor System, Federation Proc. **6**:207, 1947.

20. Bodian, D.: Experimental Evidence on the Cerebral Origin of Muscle Spasticity in Acute Poliomyelitis, Proc. Soc. Exper. Biol. & Med. **61**:170-175, 1946.

poliomyelitis would appear to require further investigation, particularly with regard to the relation of the "muscle spasm" of this disease to spasticity.[21]

A remaining question is whether injury to the reticular formation and tegmentum of the lower portion of the brain stem associated with human poliomyelitis occurs only in acutely fatal cases. The amount of material on which to base an answer is, at present, limited, but Peers's [22] recent study of 3 cases, in which the patients recovered from poliomyelitis and died of other causes, revealed in each instance a loss of neurons in the tegmentum and reticular formation of the brain stem, together with degeneration of reticulospinal tracts descending from these regions through the anterolateral portion of the cord. These observations indicate that involvement of the brain stem is not limited to fatal cases of poliomyelitis but may, with less severity, be a feature of a larger number of cases in which the outcome is not fatal.

SUMMARY

Examination of the central nervous system in a series of acutely fatal cases of poliomyelitis has revealed the distribution of severe lesions in the central core of the brain stem, with characteristic injury to the reticular formation and the tegmentum, in each instance.

The possible significance of this injury to the brain stem for the symptoms of poliomyelitis is discussed.

Northwestern University Medical School (11).

21. Magoun, H. W., and Rhines, R.: Spasticity: The Stretch Reflex and Extrapyramidal Systems, Springfield, Ill., Charles C Thomas, Publisher, 1948. Steindler, A.: The Newer Pathological and Physiological Concepts of Anterior Poliomyelitis and Their Clinical Interpretations, J. Bone & Joint Surg. **29**:59-77, 1947.

22. Peers, J. H.: The Pathology of Convalescent Poliomyelitis in Man, Am. J. Path. **19**:673-696, 1943.

PERIPHERAL NEURITIS COMPLICATING MULTIPLE MYELOMA

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IT IS well known that patients with multiple myeloma may suffer mechanical injury to neural structures as a result of compression of these tissues directly, or of their blood supply, by the tumor itself or by the pathologic fractures which it causes. Forty per cent of patients with myeloma [1] have this complication in the form of lesions of the cord,[2]

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1. (*a*) Scheinker, I.: Myelom und Nervensystem, Deutsche Ztschr. f. Nervenh. **147**:247-273, 1933. (*b*) Geschickter, C. F., and Copeland, M. M.: Multiple Myeloma, Arch. Surg. **16**:807-863 (April) 1928. (*c*) Snapper, I.: Medical Clinics in Bone Diseases, New York, Interscience Publishers, Inc., 1943, p. 182. (*d*) Antoni, N., in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 10, p. 80.

2. (*a*) Scheinker.[1a] (*b*) Rustizky, J.: Multiples Myelom, Deutsche Ztschr. f. Chir. **3**:162, 1873. (*c*) Grawitz, P.: Maligne Osteomyelitis und sarcomatöse Erkrankungen des Knochensystems als Befunde bei Fällen von perniciöser Anämie, Virchows Arch. f. path. Anat. **76**:353-364, 1879. (*d*) Seegelken, C.: Ueber multiples Myelom und Stoffwechseluntersuchungen bei demselben, Deutsches Arch. f. klin. Med. **58**:276-286, 1896-1897. (*e*) Kudrewetzky, B.: Zur Lehre von der durch Wirbelsaulentumoren bedingten Compressionserkrankung des Rückenmarkes, Ztschr. f. Heilk. **13**:300-323, 1892. (*f*) Davison, C., and Balser, B. H.: Myeloma and Its Neural Complications, Arch. Surg. **35**:913-936 (Nov.) 1937. (*g*) Jacox, H., and Kahn, E. Z.: Multiple Myeloma with Spinal Cord Involvement, Am. J. Roentgenol. **30**:201-205, 1933. (*h*) Kamman, G. R.: Solitary (?) Myeloma of the Spine with Cord Involvement, Minnesota Med. **24**:210-212, 1941. (*i*) Wright, A. D.: Solitary Plasma-Celled Myeloma of Vertebral Body, Causing Paraplegia, Proc. Roy. Soc. Med. **30**:931-932, 1937. (*j*) Winkler, K.: Das Myelom in anatomischer und klinischer Beziehung, Virchows Arch. f. path. Anat. **161**:252 and 508-528, 1900. (*k*) Wright, J. H.: A Case of Multiple Myeloma, J. Boston Soc. M. Sc. **4**:195-204, 1900. (*l*) Thomas, J. J.: A Case of Myeloma of the Spine with Compression of the Cord, Boston M. & S. J. **145**:367, 1901. (*m*) Klemme, R. M.: Plasma Cell Myelomas Causing Cord Compression: Report of Five Cases, South. M. J. **26**:692-696, 1933. (*n*) Gaube, K.: Ein Fall von multiplem Myelom unter dem Bilde einer Querschnottsmyelitis, Med. Klin. **21**:244-245, 1925. (*o*) Turner, W. G.: Myeloma of Vertebrae, J. Orthop. Surg. **19**:698, 1921. (*p*) Bertoye, H.: Contribution à l'étude de la maladie de Bence Jones, Rev. de med. **24**:390, 1904. (*q*) Bukhshtab, L. B., and Shaposhnikoff: Sur un cas de myélomatose disséminée des os du tronc avec albumosurie, Russk. Arch. patol.,

lesions of the nerve roots,[3] compression of the spinal ganglia [4] or intracranial lesions.[5] Another type of neural injury, which does not appear to be due to mechanical factors, has received little attention.

Whereas Davison and Balser [2f] were able to demonstrate pressure on neural structures or their blood supply by the myeloma or fractured vertebrae in most of their cases, the neurologic complications in 1 case could not be accounted for on this basis. In this case there was peripheral neuritis in the upper extremities with atrophy of the thenar and hypothenar eminences and interosseous muscles bilaterally. There were diminished vibratory sensation in the toes, diminished knee jerks and atrophy and fibrillations of the tongue. Degenerative changes were present in the nerve cells of the cord, and there were scattered areas of demyelination in brachial nerves. No myelomatous lesions were discovered locally to account for these changes. The authors suggested, therefore, that the lesions were "due to severe secondary anemia or unknown toxic factors." This appears to be the first description in the literature of peripheral neuritis complicating multiple myeloma. Only 1 other report of this complication has appeared. Scheinker [1a] described the case of a man aged 39 with a solitary myeloma of the sternum. Progressive palsy and paresthesia of the legs developed, followed by weakness of the right hand, with paresis of the muscles of the lower part of the arm and atrophy of the intrinsic muscles of the hand.

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klin. med. i bakt. **7**:11-21, 1899; cited by Pines and Pirogowa.[3c] (*r*) Ghon, A.: Kompressionsmyelitis durch ein Myelom, Med. Klin. **21**:74, 1925. (*s*) Flynn, J. E., and Sailer, S.: Unilateral Exophthalmos and Spinal Cord Compression in Case of Multiple Myeloma, Ohio State M. J. **37**:771-773, 1941. (*t*) Snapper, I.: On the Influence of Stilbamidine upon Multiple Myeloma, J. Mt. Sinai Hosp. **13**:119-127, 1946. (*u*) Denker, P. G., and Brock, S.: Generalized and Vertebral Forms of Myeloma: Their Cerebral and Spinal Complications, Brain **57**:291-306, 1934.

3. (*a*) Snapper.[1c] (*b*) Herz, A.: Wien. med. Wchnschr. **58**:1289, 1908. (*c*) Pines, L., and Pirogowa, L.: Ueber die multiplen Myelome und das Nervensystem, Arch. f. Psychiat. **84**:332-362, 1928.

4. von Bomhrad, H.: Ein Beitrag zum Myelom, Ztschr. f. klin. Med. **80**:506-514, 1914. Magnus-Levy, A.: Ueber die Myelomkrankheit: Beiträge zur Klinik und Pathologie, ibid. **121**:533-562, 1932.

5. (*a*) Lindeboom, G. A., and Mulder, H. J.: Multiples Myelom mit leukämischen Blutbild und degenerativen Rückenmarksänderungen, Acta med. Scandinav. **108**:363-373, 1941. (*b*) Meyer, E.: Jahreck. f. ärztl. Fortbild. **4**:100, 1913. (*c*) Denker and Brock.[2u] (*d*) Aronsohn, H. G.: Zur pathologischen Anatomie und Symptomatologie der multiplen Myelome, Virchows Arch. f. path. anat. **281**:78-87, 1931. (*e*) Kahler, O.: Zur Symptomatologie des multiplen Myeloms: Beobachtung von Albumosurie, Prag. med. Wchnschr. **14**:33 and 45, 1889. (*f*) Ritter, C.: Ueber multiple Myelome, Virchows Arch. f. path. anat. **229**:277-302, 1921. (*g*) Hammer, H.: Primäre sarcomatöse Ostitis mit chronischem Rückfallsfieber, ibid. **137**:280-317, 1894. (*h*) Anders, J. M., and Boston, L. N.: Bence-Jones Albumosuria, with Report of Three Cases: A Review of the Literature, Lancet **1**:93-97, 1903.

There ensued flaccid paralysis of the legs, paresis of both upper extremities and, finally, dyspnea, with apparent paresis of the intercostal muscles and death. The peripheral nerves revealed patchy areas of increased perineurial and epineurial connective tissue constricting the nerve bundles, with degeneration of the nerves. The distal portions of the nerves were more affected than the proximal. The spinal roots showed similar "interstitial sclerosing perineuritis." The spinal cord was normal except for degeneration of the anterior horn cells, most pronounced in the lumbar region. Scheinker expressed the belief that degeneration of the nerve cells was secondary to the peripheral neuritis. He concluded that the neurologic changes were due to toxic absorption from the solitary sternal myeloma and suggested that Bence Jones protein might be the toxin. He went so far as to suggest that the pains in various parts of the body, so characteristic of multiple myeloma, are due not to the skeletal disease but to toxins elaborated by the tumors which affect the sensory nerves.

Other authors have described neurologic complications of multiple myeloma of which postmortem examination failed to reveal adequate mechanical causes. Senator [6] described a case of bulbar palsy without any demonstrable lesion in the region of the medulla. Wieland [7] reported a case with stiff neck, slow speech, fixed pupils and mental clouding for which postmortem examination disclosed no basis. Mieremet's patient [8] had paralysis of one arm, with the tumor limited to the ilium on the same side. Examination of the spinal cord in this case revealed edema, swelling and degeneration of axis-cylinders and medullary sheaths. No vascular changes were noted.

Some authors have described cases in which mechanical compression of the spinal cord was manifest but, in their opinion, insufficient to account for the pathologic changes, for which they postulated a toxic basis.[9] Langdon [10] described the case of a woman aged 41 with multiple myeloma in which there developed palsy of the right lateral rectus

6. Senator, H.: Asthenische Lähmung Albumosurie und multiple Myelome, Berl. klin. Wchnschr. **36**:161, 1899.

7. Wieland, E.: Primär multiple Sarcome der Knochen, Bern, Jent & Co., 1893.

8. Mieremet, C. W. G.: Ueber "Systemerkrankung" und Tumorbildung der blutbereitenden Organe (zugleich ein Beitrag zur Myelomfrage), Virchows Arch. f. path. anat. **219**:1-41, 1914.

9. (*a*) Pines and Pirogowa.[3e] (*b*) Lindeboom and Mulder.[5a] (*c*) Nonne, M.: Beitrag zur Klinik der Myelom-Erkrankung, Arch. f. Dermat. u. Syph. **131**:250-256, 1921. (*d*) Kreuzer, H.: Plasma Cell Myeloma with Multiple, Partially Hemorrhagic Foci of Myelosis of Spinal Cord, Deutsche Ztschr. f. Nervenh. **90**:224-237, 1926.

10. Langdon, H. M.: Multiple Myeloma with Bilateral Sixth Nerve Paralysis and Left Retrobulbar Neuritis, Tr. Am. Ophth. Soc. **37**:223-228, 1939.

muscle, diminution in vision and atrophy of the left optic disk. In the absence of papilledema to indicate pressure on the optic nerve, the authors regarded the case as one of retrobulbar neuritis due to toxic agents. However, it must be noted that roentgenologically there were conspicuous involvement of the skull, thinning of the anterior clinoid processes and complete destruction of the posterior clinoid processes. Therefore, since no postmortem examination was made, it is illogical to exclude mechanical compression of the cranial nerves. The following cases are presented to illustrate the association of peripheral neuritis with multiple myeloma.

REPORT OF CASES

CASE 1.—J. C., a white man aged 55, was admitted because of progressive weakness, pallor and malaise, of six months' duration. He attributed these symptoms to a hernioplasty. After this operation he noted pins and needles sensations in the fourth and fifth fingers of the right hand with stiffness and weakness of the whole hand. Subsequently, he had steady aching pains in the shoulders, the right thigh, the knees and the occipital region of the head. These pains were constantly present but were worse at night and were exacerbated with motion.

Physical examination revealed extreme pallor. On both upper lids were pink, wrinkled, translucent excrescenses, about 1 cm. in diameter. There were weakness of the right hand grasp, atrophy of both thenar eminences and inability to appose either thumb. The triceps jerk was reduced on the right side. Appreciation of pain and vibratory senses was reduced in the ulnar distribution of the right hand, with pronounced hyperpathia. The knee jerks were less lively than the biceps jerks, particularly on the left side. The ankle jerks were active bilaterally. Appreciation of pain and vibratory senses was reduced in the whole right leg.

Aspiration of the sternal marrow established the diagnosis of multiple myeloma. The urine showed both Bence Jones protein and globulin. However, renal function, as measured by concentration and phenolsulfonphthalein excretion tests, was adequate. Congo red injected intravenously was not cleared from the blood. Roentgenologic examination of the skeleton revealed no arthritis or punched-out myelomatous areas. Both humeri showed decreased calcification.

The patient was treated with "stilbamidine" (4,4-diaminostilbene),[11] with notable increase in mobility of the shoulders and limbs and with loss of pain. No objective neurologic improvement was seen.

CASE 2.—F. K., a white woman aged 53, was admitted to the hospital in June 1946 with the chief complaint of pain in the lower part of the back, of nine months' duration, and paralysis of the lower limbs, of three months' duration. She had been well until the year prior to admission, when she noted gradually increasing fatigue of the legs. This was followed by progressively severe pain in the lower part of the back and pain in the lower limbs, involving first the thighs and later the knees, legs and ankles. She was examined at the Neurological Institute of New York, where roentgenograms demonstrated fracture of the first lumbar vertebra. Biopsy showed a plasma cell myeloma. Shortly after operation, the paresis of the legs progressed to flaccid paralysis, and at this time she lost partial control of both hands. Bladder and bowel control remained intact. There was intense pain in the lower portions of the legs and the feet.

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11. Snapper, T.: Stilbamidine and Pentamidine in Multiple Myeloma, J. A. M. A. **133**:157-161 (Jan. 18) 1947; footnote 2 *t*.

Physical examination revealed that the patient was obese and well developed. There was bilateral foot drop with paralysis of the extensor and flexor muscles of the feet and toes. Power in the knees and hips was fairly good. There was severe weakness of the fingers and of the flexors of the wrists, with well marked clawhand bilaterally and atrophy of the intrinsic muscles of the hands. The right latissimus dorsi muscle did not contract on coughing, and there was weakness of elevation of the right shoulder. All deep reflexes except the jaw jerk were absent. Touch and position senses were absent bilaterally in the stocking area, with absence of vibratory sense to the iliac crests. Position sense was faulty in the fingers, with loss of fine motion and of astereognosis in the hands.

Aspiration of sternal marrow confirmed the diagnosis of multiple myeloma. A roentgenogram again showed involvement of the first lumbar vertebra without compression and demonstrated lesions in the vault of the skull. The rest of the spine, the pelvis, the clavicles and the long bones appeared normal. Bence Jones protein was not present. Lumbar puncture in the third interspace showed no block, and the cytologic and chemical constituents of the spinal fluid were normal.

The patient was treated with "stilbamidine" and thiamine hydrochloride, with relief of pain but with no change in the neurologic signs.

CASE 3.—G. E., a white man aged 59, was first admitted to the hospital in June 1945 with the chief complaint of pain in the legs of six weeks' duration. The pain was chiefly nocturnal, beginning in the calves and radiating to the toes. It was relieved with acetylsalicylic acid U. S. P. Diabetes mellitus of mild degree had been present for twenty-four years.

At the first admission physical examination revealed only pallor and tenderness in the muscles of the lower extremities. Normochromic anemia, with a hemoglobin concentration of 40 per cent, was present. The leukocyte count was normal. The stools gave a positive reaction to the guaiac test. The erythrocyte sedimentation rate was 123 mm. per hour (Westergren method). Aspiration of the bone marrow revealed general hypoplasia. A barium sulfate meal was interpreted as showing a duodenal ulcer, and the syndrome was interpreted as chronic loss of blood due to the peptic ulcer and diabetes mellitus with early neuropathy.

In May 1946 the patient returned, complaining of two types of pain. One, of eleven months' duration, was a progressively severe hyperesthesia of the abdominal wall, which, in the two months prior to his admission, had become an intolerable burning sensation on friction with clothing; the second, of two months' duration, was an aching nocturnal soreness in the thighs and legs, a sensation "as of being beaten with a stick."

Examination at this time revealed pallor with intense hyperesthesia of the feet. Position sense was normal, but vibration sense was slightly reduced. In a zone extending from the nipple line to the groin definite hyperaphia was noted. There was general hyporeflexia, especially noticeable in the achilles jerk. The abdominal reflexes were lively.

Roentgenologic examination demonstrated compression fractures of the tenth, eleventh and twelfth thoracic vertebrae and partial compression of the seventh, eighth and ninth thoracic vertebrae. The sternal marrow was characteristic of multiple myeloma. Bence Jones proteinuria was not present.

The patient was treated with "stilbamidine" and thiamine hydrochloride (50 mg. daily), with progressive increase in strength, subsidence of the pain in the legs and complete clearing of the cutaneous hyperesthesia.

The third admission, in September 1946, was for diabetic coma. The sensory status could not be determined. The ankle and knee jerks were absent. The

bladder was apparently atonic, but because of the coma the mechanism for this could not be determined. Despite therapy, the patient died on the second day in the hospital.

Postmortem examination revealed only minor involvement of bone with small nodules in the sternum, the twelfth dorsal and the third lumbar vertebrae and the iliac bones. The spinal cord was intact. The peripheral nerves were not examined.

COMMENT

In our first 2 cases, the skeletal involvement by the myeloma was minimal, as disclosed roentgenologically, and the principal symptoms were due to the neuritis. It is impossible to exclude compression of the cord and nerve roots by tumor, but the lack of evidence of lesions of the vertebrae corresponding to the level of the neurologic involvement and the normal spinal fluid do not favor this possibility. The peripheral type of lesion in the upper extremities, characterized by sensory and motor changes and by muscular atrophy, closely resembles that in Scheinker's [1a] and in Davison and Balser's case.[2f] As has been pointed out, the localization of the myeloma to the sternum in Scheinker's case excludes any possibility of mechanical injury to neural structures. The similarity between this case and our cases suggests that the neuritis in ours was in all probablity also toxic.

The peripheral type of neural lesion encountered in our third case cannot definitely be ascribed to the multiple myeloma in view of the long-standing mild diabetes. The bizarre symptoms suggest, however, that the neuropathy in this case may also have been secondary to the myeloma.

Scheinker suggested that the neurologic changes are due to absorption of toxins from the myeloma. He expressed the belief that this toxic product might be Bence Jones protein. However, as has been noted, in cases 2 and 3 in our series the Bence Jones proteinuria was not present. Kreuzer [9d] suggested that the toxins elaborated by the tumor cells result in the production of Bence Jones protein, amyloidosis and asthenia. Our observations do not permit us to draw any conclusions as to the pathogenesis of the peripheral neuritis occasionally observed with multiple myeloma. We wish only to draw attention to the fact that multiple myeloma, in common with some metabolic diseases such as diabetes and pernicious anemia, can give rise to a neuropathy independently of its well known mechanical effects.

SUMMARY

The literature on neural complications of multiple myeloma is reviewed. About 40 per cent of patients with myeloma have this complication. Only 2 cases of peripheral neuritis have previously been reported.

Three cases of peripheral neuritis are presented. In 1 of these the myeloma was complicated by incidental diabetes.

No conclusions as to the pathogenesis of this "toxic" neuritis could be drawn. However, attention is invited to the existence of this little recognized syndrome.

Rockefeller Institute for Medical Research.

INVOLUTIONAL PSYCHOSIS

Course When Untreated and When Treated with Electric Shock

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IN THIS report are compared the courses of illness of two series of patients with involutional psychoses, one treated with electric shock and the other not so treated. An analysis of certain factors possibly related to the course is also presented.

Involutional psychosis is a psychosis occurring in middle life and the following years, without evidence of either "organic" intellectual defects or a previous affective disorder. The subtype melancholia is characterized mainly by agitation and depression, with a mental content of self condemnation, hopelessness and a tendency toward hypochondriasis. The paranoid subtype shows delusions of persecution or grandiosity, suspiciousness and misinterpretations. The mixed subtype is a combination of these two types, with the addition of strong somatic delusions in some cases.

MATERIALS AND TECHNIC

The control group consisted of patients hospitalized from 1930 to 1939, inclusive, and given hydrotherapy, psychotherapy, participation in group activities and occupational therapy. None received any form of shock therapy. The electric shock group was treated in the hospital from July 1, 1941 to July 1, 1943. For the control group, follow-up information was obtained, by means of letter, for periods up to fifteen years from the patients, relatives, social agencies, referring physicians, local and state hospitals, county homes and private sanatoriums. Also, we examined one third of the patients in the outpatient clinic, in their homes and in state hospitals.[1] In the case of death from uncertain causes, a report was secured from the State Bureau of Vital Statistics. No patient was included unless the follow-up information was considered reliable. On the average, three sources of information were checked for each patient. The shock group was followed by the aforementioned technics every six months after discharge up to a maximum of four years.

The control group comprised 93 patients, 60 women and 33 men, with a condition diagnosed as involutional melancholia. Of the 60 women, 45 were

1. Permission to examine patients and records in state hospitals was obtained from Dr. R. A. Stewart, Independence State Hospital; Dr. C. F. Obermann, Cherokee State Hospital; Dr. N. Render, Clarinda State Hospital, and Dr. A. Soucek, Mount Pleasant State Hospital.

classified as having a typical illness and 15 as having an atypical illness. Of the 33 men, 23 had a typical and 10 an atypical illness. A typical illness was one with agitated depression. An atypical illness was one with mild paranoid or somatic delusions or with pronounced retardation. The atypical features, however, were not of sufficiently severe degree to remove the patients from the category of the melancholia subtype.

The shock group included 61 patients, classified according to the following subtypes: melancholia, 50 patients; paranoia, 5 patients, and mixed, 6 patients. The melancholia group contained 33 women and 17 men; the paranoid group was composed entirely of women, and in the mixed group there were 4 women and 2 men.

Table 1 presents the statistics with respect to age of onset of the psychosis, duration of psychosis before admission and length of follow-up period. The patients of the control and the shock groups were of about the same age at the onset of the psychosis, 52 and 50 years, respectively. The patients of both groups, too, had been ill from six to seven months before admission. The median follow-up period for the control group, of seventy-eight months, was forty-two months longer than the median period, of thirty-six months, for the shock group.

TABLE 1.—*Statistical Data on Age of Onset, Duration of Illness Before Admission, and Length of Follow-Up Period for Patients with Involutional Psychosis*

	Control Group					Shock Group				
	No.	Mean	Median	Middle 50%	Range	No.	Mean	Median	Middle 50%	Range
Age of onset (years)										
Men	33	53.2	53	48-57	40-72	19	52	52	49-54	39-63
Women	60	50.7	50	46-54	36-67	42	50.3	49	44-55	37-70
Total	93	51.6	52	48-56	36-72	61	50.8	50	46-56	37-70
Duration of illness before admission (months)	93	10	6	3-12	½-72	61	9.9	7	4-12	1-72
Follow-up period (months)	93	77	78	36-120	1/15-180	61	35.6	36	30-42	1-48

Extremely short follow-up periods represent patients who committed suicide or died soon after discharge. Since the patients of the two groups were fairly comparable with respect to age of onset and duration of the psychosis before admission, it seems legitimate to compare their subsequent courses. The two groups were drawn from the same cultural and racial populations.

Forty-six of the patients in the shock group received one series of shocks; 13 patients, two series; 1 patient, three series, and 1 patient, six series. Multiple series were given to some of the patients who relapsed. For the patients who received only one series, the average number of shocks per patient was eight: the median number of shocks, seven; the middle 50 per cent, six to ten shocks, and the range, two to sixteen. The average number of shocks for the patients receiving more than one series was seventeen. By shock is meant a major convulsion. From two to three shock treatments a week were given. They were produced with a 60 cycle current applied from 0.1 to 0.3 second with currents of 80 to 150 volts. Curare was injected intravenously before most of the treatments to weaken the convulsions. After recovery had occurred, one or two more convulsions were induced. If recovery did not occur, shock therapy was continued up to a total of ten to sixteen treatments. There were no complications with shock other than transient loss of memory or occasional temporary postshock confusion.

RESULTS

Outcome and Duration.—Control Group: The average duration of the illness for all 93 patients was fifty-four months, with a median duration of thirty-seven months, a middle 50 per cent of fifteen to eighty-one months and a range of two to two hundred and four months. These figures, however, obscure important details, which are presented in table 2. Here, the data are separated into four follow-up categories: complete recovery, illness still present, suicide and death from causes other than suicide.

TABLE 2.—*Duration (in Months) of Untreated Involutional Melancholia According to Categories of Outcome*

	No.	Mean	Median	Middle 50%	Range
Whole group					
Men	33	69	51	8-115	2-204
Women	60	46	30	14- 68	3-186
Total	93 (100%)	54	37	15- 81	2-204
Complete recovery					
Men	16	53	30	15-108	7-138
Women	27	46	32	17- 66	8-145
Total	43 (46%)	49	31	15- 66	7-145
Illness still present					
Men	8	136	130	111-157	81-204
Women	9	96	115	81-144	49-186
Total	17 (18%)	115	115	81-144	49-204
Suicide					
Men	4	54	61	55- 67	15- 78
Women	8	17	15	11- 23	10- 26
Total	12 (13%)	29	15	15- 26	10- 78
Death					
Men	5	23	24	13- 27	2- 51
Women	16	32	18	18- 44	3-108
Total	21 (23%)	30	18	8- 44	2-108

Table 2 shows that 43 patients, or 46 per cent of the total group, are completely recovered. In 17, or 18 per cent, the illness is still present. Twelve, or 13 per cent, committed suicide; and 21, or 23 per cent, are dead. For the category of complete recovery, the average duration of illness was forty-nine months; the median duration, thirty-one months; the middle 50 per cent, fifteen to sixty-six months, and the range, seven to one hundred and forty-five months. The average and median figures (table 2) for the category of illness still present were much greater than those for the category of complete recovery. Three fourths of the patients who committed suicide did so within a period of about two years after the onset. Of the 21 deaths, 9 occurred as a direct result of the mental illness. These patients died of exhaustion or malnutrition because of extreme agitation or refusal to eat. Of the

other 12 deaths, cardiovascular disease was the cause of 5; pneumonia, of 2; leukemia, of 1; ulcerative colitis, of 1; osteogenic sarcoma, of 1, and the cause of 1 death was unknown.

Electric Shock Group: Table 3 shows the course for follow-up periods of six months according to the response categories of complete recovery, pronounced improvement, improvement and no improvement. From 75 to 85 per cent of the group were in the categories of complete recovery and pronounced improvement throughout the whole follow-up period of forty-eight months. More patients were in the category of complete recovery than in that of pronounced improvement throughout the period of observation, except at discharge; the latter may represent the conservatism of the staff in judging the status at discharge.

Table 4 presents the course according to diagnostic subgroups. Here, the categories of complete recovery and pronounced improvement

TABLE 3.—*Follow-Up Course After Electric Shock Therapy of 61 Patients with Involutional Psychoses*

	At Discharge, No.	Follow-Up Period (Months)							
		6 No.	12 No.	18 No.	24 No.	30 No.	36 No.	42 No.	48 No.
Complete recovery.....	22 (77%)	32 (75%)	39 (78%)	39 (78%)	37 (76%)	29 (85%)	26 (85%)	16 (93%)	3 (80%)
Pronounced improvement...	25	13	8	8	6	11	6	4	1
Improvement.....	12 (23%)	4 (25%)	6 (22%)	6 (22%)	7 (24%)	1 (15%)	2 (11%)	1 (7%)	0 (20%)
No improvement.....	2	11	7	7	7	6	2	1	1
Toal no. of patients....	61	60	60	60	57	47	36	22	5

have been combined, as have the categories of improvement and no improvement. The responses of the melancholia and the mixed subgroup are about the same, and the responses of both are better than those of the paranoid group. To check on the last point, the patients in the mixed subgroup with paranoid features were combined with the paranoid group. This emphasizes the relatively poor response of a psychosis with paranoid features (table 4).

A more detailed picture is obtained from an analysis of the individual cases. Thirty-four of the 61 patients were at a level of complete recovery for most of the follow-up period. Two patients were graded as showing pronounced improvement and 2 as unimproved throughout. Four patients who failed to respond were given a second series of treatments at a later date, and 3 completely recovered. Two patients who did not respond recovered spontaneously later. One patient who did not respond committed suicide one month after discharge. Sixteen patients, or 31 per cent, relapsed after shock therapy. The average time of

relapse after treatment was ten months; the median period, six months, and the range, one week to forty-eight months. Eleven of these patients received more shock therapy. Nine were given a second series; 8 of these responded with complete recovery and have remained well for an average period of twenty-two months; the other patient responded but relapsed after one year; 1 patient received two more series, responded with the first, then relapsed and failed to respond to the last series, and 1 patient received five more series in the two years after the first series, making a total of fifty shocks. The last patient reached a level

TABLE 4.—*Follow-Up Course of Involutional Psychoses After Electric Shock Therapy According to Diagnostic Subtypes*

Outcome Category	At Discharge, No.	Follow-Up Period (Months) 6 No.	12 No.	18 No.	24 No.	30 No.	36 No.	42 No.	48 No.
Involutional Melancholia (50 Patients)									
Complete recovery and pronounced improvement...	42 (84%)	39 (78%)	42 (84%)	41 (82%)	37 (77%)	37 (88%)	28 (90%)	17 (95%)	4 (100%)
Improvement and no improvement.....	8 (16%)	11 (22%)	8 (16%)	9 (18%)	11 (23%)	5 (12%)	3 (10%)	1 (5%)	
Involutional Psychosis, Mixed Type (6 Patients)									
Complete recovery and pronounced improvement...	4 (67%)	4 (80%)	4 (80%)	4 (80%)	4 (80%)	2 (100%)	2 (100%)	2 (100%)	
Improvement and no improvement.....	2 (33%)	1 (20%)	1 (20%)	1 (20%)	1 (20%)				
Involutional Psychosis, Paranoid Type (5 Patients)									
Complete recovery and pronounced improvement...	1 (20%)	2 (40%)	2 40%)	2 (40%)	2 (50%)	1 (33%)	2 (67%)	2 (67%)	
Improvement and no improvement.....	4 (80%)	3 (60%)	3 (60%)	3 (60%)	2 (50%)	2 (67%)	1 (33%)	1 (33%)	1 (100%)
Involutional Psychosis, Paranoid Type and Mixed Type with Paranoid Features (8 Patients)									
Complete recovery and pronounced improvement...	2 (25%)	3 (43%)	3 (43%)	3 (43%)	3 (50%)	2 (50%)	3 (60%)	3 (60%)	3 (60%)
Improvement and no improvement.....	6 (75%)	4 (57%)	4 (57%)	4 (57%)	3 (50%)	2 (50%)	2 (40%)	2 (40%)	2 (40%)

of pronounced improvement after the first five series, only to relapse each time after a few months. She attained complete recovery with the sixth series and has held this status for a year.

Time Spent in Psychiatric Hospitals.—Control Group: The 93 patients stayed in psychiatric hospitals an average of twenty-one months, with a median of seven months, a middle 50 per cent of one to twenty-three months and a range of one-half to one hundred and eighty months. The men were hospitalized longer than the women, the mean for the men being thirty-one months, the median nine months, the middle 50 per cent three to thirty-seven months and the range one to one hundred and eighty months, whereas the corresponding figures for the women

were fifteen, five, one to seventeen and one-half to one hundred and eight months.

Shock Group: The mean residence in hospital was six months; the median, one and one-half months; the middle 50 per cent, one to two and one-half months, and the range, three-fourths to forty months. The mean for the shock group was high because 3 patients were included who did not respond to shock therapy and were transferred to state hospitals. Patients who responded to shock were usually in the hospital from four to five weeks.[2]

FACTORS RELATED TO COURSE

In this section is presented the relation of certain factors to the courses of illness of the untreated and the treated patients.[3] It was

2. (*a*) Alexander, G. H.: Therapeutic Efficacy of Electroconvulsive Therapy: Comparative Classification of Treatment Results Determined With and Without Use of Time Factor in Their Evaluations, J. Nerv. & Ment. Dis. **102**:221-230, 1945. (*b*) Batt, J. C.: One Hundred Depressive Psychoses Treated with Electrically Induced Convulsions, J. Ment. Sc. **89**:289-296, 1943. (*c*) Bennett, A. E.: An Evaluation of the Shock Therapies, Psychiatric Quart. **19**:465-477, 1945. (*d*) Bianchi, J. A., and Chiarello, C. J.: Shock Therapy in the Involutional and Manic-Depressive Psychoses, ibid. **18**:118-126, 1944. (*e*) Davidoff, E., and Raffaele, A.: Electric Shock Therapy in Involutional Psychoses, J. Nerv. & Ment. Dis. **99**:397-405, 1944. (*f*) Drobnes, S.: Prognostic Factors in the Involutional Psychoses, Am. J. Psychiat. **99**:818-821, 1943. (*g*) Epstein, J.: Electric Shock Therapy in the Psychoses: Study of One Hundred Cases, J. Nerv. & Ment. Dis. **98**:115-129, 1943. (*h*) Furst, W., and Stouffer, J. F.: The Electrical Shock Treatment of Psychoses, ibid. **96**:499-507, 1942. (*i*) Kalinowsky, L. B., and Hoch, P. H.: Shock Treatments and Other Somatic Procedures in Psychiatry, New York, Grune & Stratton, Inc., 1946. (*j*) Kalinowsky, L. B.: Electric Convulsive Therapy with Emphasis on Importance of Adequate Treatment, Arch. Neurol & Psychiat. **50**:652-660 (Dec.) 1943. (*k*) Katzenelbogen, S.; Baur, A. K., and Coyne, A. R. M.: Electric Shock Therapy: Clinical, Biochemical and Morphologic Studies, ibid. **52**:323-326 (Oct.) 1944. (*l*) Kerman, E. F.: Electroshock Therapy, with Special Reference to Relapses and Effort to Prevent Them, J. Nerv. & Ment. Dis. **102**:231-242, 1945. (*m*) Malzberg, B.: Outcome of Electric Shock Therapy in the New York Civil State Hospitals, Psychiatric Quart. **17**:154-163, 1943. (*n*) Palmer, H. D., and Sherman, S. H.: The Involutional Melancholia Process, Arch. Neurol. & Psychiat. **40**:762-788 (Oct.) 1938. (*o*) Palmer, H. D.; Hastings, D. W., and Sherman, S. H.: Therapy in Involutional Melancholia, Am. J. Psychiat. **97**:1086-1115, 1941. (*p*) Smith, L. H.; Hughes, J.; Hastings, D. W., and Alpers, B. J.: Electroshock Treatment in the Psychoses, ibid. **98**:558-561, 1942. (*q*) Smith, L. H.; Hastings, D. W., and Hughes, J.: Immediate and Follow-Up Results of Electroshock Therapy, ibid. **100**:351-354, 1943. (*r*) Taylor, J. H.: Involutional Psychoses Treated with Shock Therapy, Dis. Nerv. System **6**:189-190, 1945. (*s*) Tillotson, K. J., and Sulzbach, W.: A Comparative Study and Evaluation of Electric Shock Therapy in Depressive States, Am. J. Psychiat. **101**:455-459, 1945. (*t*) Ziskind, E.; Somerfeld-Ziskind, E., and Ziskind, L.: Metrazol and Electric Convulsive Therapy of the Affective Psychoses: Controlled Series of Observations Covering Period of Five Years, Arch. Neurol. & Psychiat. **53**:212-217 (March) 1945.

3. Drobnes.[2f] Palmer and Sherman.[2n] Palmer, Hastings and Sherman.[2o]

hoped that some of these factors might have prognostic value. The factors were: family history, age of onset of the psychosis, personality type, type of onset, acuteness of onset, severity of symptoms, typicality of the psychosis, duration of illness before treatment, number of shocks and sex.

Family History.—The family histories of the groups were analyzed for psychosis and for instability in the immediate family and in the collateral lines for the two preceding generations. By psychosis was meant a manic-depressive, involutional or schizophrenic illness; by instability, psychopathy, alcoholism, criminality, epilepsy, or "nervous breakdown." No relation was found between family history and the outcome or duration of the psychosis in the control group, or between family history and the response to shock therapy.

Age of Onset of Psychosis.—No relation was found between the age of onset and the duration of illness in the category of complete recovery in the control group. Nor was there any association between age of onset and the response to shock therapy.

Personality Type.—In the control group an unequal distribution of personality types made analysis unprofitable. In the shock group 50 per cent of the patients had normal personalities prior to the onset of the psychosis and 38 per cent had anancastic personalities. No remarkable difference was noted in the response to treatment.

Type of Onset.—The two groups of patients were divided into those with illnesses of endogenous, psychogenic and organogenic onsets. An endogenous onset was one in which no known precipitating factor was present; a psychogenic onset, one in which some emotional trauma occurred, and an organogenic onset, one in which some physical illness preceded the mental illness. In the control group the outcome and duration of the psychosis were about the same for each type of onset except that there was a greater proportion of deaths (other than suicide) for the organogenic group and for a combined psychogenic and organogenic group. Fifty per cent of the 12 patients with organogenic onset died, while only 19 per cent (15 out of 81) of the patients having psychogenic and endogenous onsets met a similar fate. In the shock group the type of onset was without significant influence on the results of shock.

Acuteness of Onset.—Eighteen of the control patients had acute onset of the illness, i. e., development in less than one month; 75 patients had a gradual onset. The duration and outcome were the same for the two groups. Of the 50 patients with melancholia who received shock therapy, the onset was acute in 10 and gradual in 40. The response to treatment was similar regardless of the type of onset.

Severity of Symptoms.—For the control group, severity of symptoms (severe in 37 patients, moderate in 43 patients and mild in 13 patients) was not related to the percentage of patients in the four follow-up categories, nor was there any effect on the duration of the illness. However, severer symptoms may be related to early suicide and death. Thus, the median duration before suicide for patients with severe symptoms was ten months (3 patients), for those with moderate symptoms, twenty-five months (8 patients) and for those with mild symptoms, fifty-five months (1 patient). The median duration before death for patients with severe symptoms was fourteen months (13 patients), and for those with moderate symptoms, thirty-nine months (8 patients). The response throughout the follow-up period of the 50 patients with melancholia who had received therapy was the same regardless of the severity of the symptoms.

Typicality of Psychosis.—Of the patients with atypical psychoses in the control group, 9 showed paranoid features and 3 somatic features; 2 had mixtures of these, and 11 were severely retarded. The outcome and duration of the illness for these patients were about the same as for the 68 patients with typical agitated depressions. In the shock group the poorer response of patients with paranoid features has been mentioned (table 4).

Duration of Illness Before Treatment.—In the shock group the duration of illness before treatment was divided into three month intervals. The course after therapy was approximately the same for all durations. Apparently, late treatment is as effective as early treatment.

Number of Shocks.—No relation could be demonstrated between the number of shocks and the subsequent course of the illness. There is no specific number of shocks in treatment of the psychosis. Treatment in each case must be individualized.

Sex.—After the control and shock groups were divided according to sex, the course was studied with respect to family history, age of onset, personality type, type of onset, acuteness of onset, severity of symptoms, duration of illness before treatment and number of shocks. No sex differences were apparent in either the control or the shock group.

Patients Who Failed to Respond or Who Relapsed.—An analysis was made of the data for the shocked patients who did not respond with complete recovery or pronounced improvement. This analysis involved age of onset, type of onset (endogenous, psychogenic), acuteness of onset, premorbid personality, severity of symptoms, family history and sex. No difference in the distribution of these factors was present when this group was compared with the patients who responded favorably. A similar analysis for patients who relapsed showed no positive results with respect to these factors.

COMMENT

In a comparison of the courses of the untreated and the treated group the figures speak for themselves. An illness from which only about one-half the patients recovered spontaneously, whereas one-fifth remained ill, one-tenth committed suicide, one-tenth died directly as a result of the illness and one-tenth died of other causes, now shows a favorable response to electric shock in four fifths of the patients. Electric shock, furthermore, has shortened notably the duration of the illness and the time of hospital residence.

Failure to respond to, or a relapse after, one series of shock treatments does not indicate a hopeless prognosis. Three fourths of our patients who failed to respond to one series did so after one or more additional series at some later date. Likewise, of the 31 per cent of patients who relapsed after one series three-fourths were treated again, and 80 per cent responded with one or more series. We can give no reason that some patients failed to respond or relapsed. Nor can we explain why some patients required two or more series for a favorable response. It might be urged that those who responded finally were near the end of the natural course of their psychosis. However, their recovery coincided with a subsequent shock series. Concerning the one tenth of the control group who died of causes not directly the result of the mental illness, it is interesting that 5 of these deaths were cardiovascular. Chronic agitation may place a severe strain on the vascular system. Plausibility is added to this suggestion by the absence of similar deaths in the shock group in a comparable follow-up period.

In the control group the failure to find any significant relation between the course and the factors of family history, age of onset of the psychosis, acuteness of onset, typicality of the psychosis and sex apparently means that other factors, as yet unknown, are of greater importance. The relation between severe symptoms and early suicide or death in the control group is of practical value in advising early treatment. Similar advice might be considered in the case of patients with "organic" onsets, since a greater proportion of deaths occurred among these patients. Likewise, the absence of significant correlations between the results of follow-up observation in the shock group and the factors of family history, age of onset, type of personality, type of onset, acuteness of onset, severity of symptoms, duration of illness before treatment, number of shocks and sex is of both practical and theoretic value. Practically, it can be said that none of these factors appears to influence the results of shock. The theoretic value is a negative one in that these factors may be excluded probably in further attempts to understand the mechanism of shock therapy.

SUMMARY AND CONCLUSIONS

The courses of two groups of patients with involutional psychosis, a control group of 93 and a group of 61 treated by electric shock, are compared. The control group was followed for periods up to fifteen years, with an average of six and one-half years, after discharge; and the shock group, up to four years, with an average of three years.

In the control group, 46 per cent of the patients recovered spontaneously after a median duration of illness of thirty-one months; 18 per cent of the patients are still ill; 13 per cent committed suicide, and 23 per cent died. About one-half these deaths were directly attributable to the mental illness. In the shock group, 80 per cent of the patients were completely recovered or greatly improved for the whole follow-up period. The patients with the paranoid form showed a much poorer response than the patients with melancholia. A few patients who failed to respond to shock therapy did so when a second series was given later. Sixteen patients, or 31 per cent, relapsed after the first series of shock, and 11 of these received additional treatment; 80 per cent again responded favorably. Shock treatment reduced the residence in hospital from a median of seven months to a median of one and one-half months.

In the control group, it was indicated that the following factors had no important relation to the course: a positive family history of psychosis or instability, age of onset of the psychosis, acuteness of onset, sex and typicality of the psychosis. There was evidence that patients with organic factors in the onset died sooner than those with a psychogenic or an endogenous onset, and that patients with severe symptoms committed suicide and died sooner after the onset than did those with mild symptoms.

In the shock group, it was indicated that the following factors have little or no influence on the results of treatment: a positive family history of psychosis or instability, age of onset, premorbid personality characteristics, type of onset (endogenous, psychogenic), acuteness of onset, severity of symptoms, duration of illness before treatment, number of shocks and sex.

No factors could be found to characterize the patients who failed to respond to shock or who relapsed after treatment.

It is concluded that electric shock therapy should be applied early to patients with involutional psychoses, and should be repeated if failure or relapse occurs. It is also concluded that the factors analyzed for a possible relation to the results of shock can be neglected, in general, in efforts to understand the mechanism of shock therapy.

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THE ELECTROENCEPHALOGRAM IN CEREBROVASCULAR DISEASE

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A SURVEY of the literature yields little information on the electroencephalogram of patients with cerebrovascular disease (cerebral thrombosis, cerebral hemorrhage and cerebral embolism). Although Gibbs and Gibbs, in their "Atlas of Electroencephalography," [1] reported 1 case each of cerebral arteriosclerosis, cerebral embolism and multiple cerebral hemorrhage, we found no publication dealing specifically with the electroencephalograms of this important group of patients. Because of this fact, and also because such patients so frequently present a problem of differential diagnosis from expanding intracranial lesions, the electroencephalograms of 95 patients with a disturbance diagnosed as cerebrovascular disease were studied. The characteristics of these records were determined; their possible use as an aid in differential diagnosis was investigated, and an attempt was made to correlate the electroencephalogram with the clinical findings.

TECHNIC

Standard records were taken with a three channel Grass electroencephalograph, using eleven electrodes, as follows: one vertex electrode (placed in the midline, halfway between the nasion and the inion); two "indifferent" electrodes (placed on the right and the left ear lobe); four pairs of symmetric electrodes above the hemispheres, namely, 1 pair of frontal electrodes (placed halfway between the nasion and the vertex and 3 cm. on either side of the midline), 1 pair of central electrodes (placed 3 cm. lateral to the vertex electrode), 1 pair of parietal electrodes (placed 4 cm. behind the vertex and 7 cm. on either side of the midline) and 1 pair of occipital electrodes (placed 3 cm. in front of the inion and 3 cm. on either side of the midline).

Records from symmetric electrodes were taken simultaneously, using the following leads: Frontal-occipital, frontal-vertex, central-vertex, parietal-vertex, occipital-vertex, frontal-indifferent, central-indifferent, parietal-indifferent, occipital-indifferent, frontal-central, central-parietal and parietal-occipital.

From the Neurological Service (Dr. Israel S. Wechsler, Director), of the Mount Sinai Hospital.

1. Gibbs, F. A., and Gibbs, E. L.: Atlas of Electroencephalography, Cambridge, Mass., Lew A. Cummings, 1941, pp. 152-155.

With some modifications, the records were classified according to the method previously described by one of us (H. S.).[2] In describing this classification, the following terms should be defined: delta index; average delta index; delta quotient; high degree of abnormality (H); low degree of abnormality (L); symmetric record (S); asymmetric (A) and parasymmetric (P) records, and the letters *D*, *F* and *B*. The delta index is the percentage of total length of record occupied by delta activity, i. e., waves of a frequency of 6 or less per second and a voltage of at least 20 microvolts. The average delta index is the value obtained by adding the delta indexes of all the leads used and dividing the sum by the number of leads. The delta quotient is the figure obtained by dividing the sum of all the delta index values for the more abnormal side by the corresponding sum for the other side. A record with a high degree of abnormality is one in which the lowest frequency is 2 cycles or less per second, the average delta index 20 or more and the delta index 35 or more in at least one lead. A record with a low degree of abnormality is one in which any one of these conditions is not fulfilled. A symmetric record is one in which the delta activity is the same on the two sides with respect to frequency, voltage and time of appearance. Records with a high degree of abnormality are considered asymmetric when the delta quotient is greater than 1.5 and as parasymmetric when the delta quotient is 1.5 or less. Records with a low degree of abnormality are considered asymmetric when the delta quotient is greater than 2 and as parasymmetric when the delta quotient is 2 or less. The letter *D* indicates a diffusely abnormal record without an electroencephalographic focus. The letter *F* indicates a record with such a focus. The letter *B* denotes bursts of three or more delta waves having the same frequency and amplitude and sharply delimited as a group.

RESULTS

Cerebrovascular Disease with no Delta Activity.—Of the 95 patients, 67 had records with no delta activity. These records were taken at intervals varying from one day to one year after the onset of illness. No patient was mentally clouded at the time of recording. The clinical diagnoses for these patients were as follows: Sixty-five patients had cerebral arteriosclerosis and cerebral thrombosis, and 2 patients, cerebral embolism. Forty-four patients having normal electroencephalograms showed clinical signs of severe hemispheric lesions, such as hemiparesis (24 patients), hemiparesis with aphasia (8 patients), hemiparesis with hemisensory defects (3 patients), hemianopsia (6 patients), hemianopsia with aphasia (1 patient) and aphasia (2 patients). There were 4 patients with headache and dizziness, 3 with psychosis, 5 with personality changes and memory defects and 11 with a brain stem syndrome, including 6 with a syndrome of involvement of the posterior inferior cerebellar artery.

For 2 patients with severe focal signs of hemispheric lesions electroencephalograms were taken one day after the onset of symptoms,

2. Strauss, H.: Electroencephalographic Studies: A Method for the Differential Diagnosis of Abnormal Electroencephalograms, J. Mt. Sinai Hosp. **9**:17-24, 1942.

and both had normal records. Thirteen of the patients had epileptic attacks, which were generalized in 9 and jacksonian in 4. In no instance did the electroencephalogram show any abnormality suggesting a convulsive disorder.

Cerebrovascular Disease with a Low Degree of Electroencephalographic Abnormality of Symmetric, Diffuse Type (LS-D).—Of the 95 patients, 8 showed a low degree of electroencephalographic abnormality of symmetric, diffuse type. Two patients with a condition diagnosed as cerebral thrombosis were drowsy at the time the electroencephalograms were recorded. On 1 of these 2 patients, the records were taken on the fifth day of illness and did not show any focal symptoms. The other patient presented a syndrome referable to the brain stem. For the latter the electroencephalogram was recorded one day after the onset of symptoms. A second record taken three days later, at which time the patient was mentally alert, showed a lessening in the degree of abnormality. For the remaining 6 patients the clinical diagnosis included cerebral arteriosclerosis and thrombosis (5 patients) and cerebral embolism (1 patient). The electroencephalograms of these 6 patients were recorded at intervals varying from three days to one year after the onset of symptoms. The clinical symptoms and signs of the 6 patients without any clouding of consciousness included hemiparesis (1 patient), hemiparesis with aphasia (1 patient), headache and dizziness (3 patients) and personality changes and memory defects (1 patient).

Cerebrovascular Disease with a Low Degree of Electroencephalographic Abnormality of Asymmetric, Focal Type (LA-F).—Three patients had records of this type. All were mentally alert. The electroencephalograms were recorded at intervals of eight days to one year after the onset of illness. The clinical symptoms included hemiparesis with aphasia (2 patients) and hemiparesis with hemisensory defect (1 patient). In all 3 patients the electroencephalographic focus corresponded to the focus postulated on the basis of the clinical status.

Cerebrovascular Disease with a Low Degree of Electroencephalographic Abnormality of Parasymmetric, Focal Type (LP-F).—Only 1 patient had a record of this type. The electroencephalogram was taken six hours after the onset of symptoms. The patient was mentally alert. The clinical diagnosis was cerebral embolism, and the clinical symptoms included hemiparesis with aphasia. Here, too, the electroencephalograpic focus corresponded to the focus postulated on the basis of the clinical status. A repeat record taken three days later showed diminution in the degree of abnormality.

Cerebrovascular Disease with a High Degree of Electroencephalographic Abnormality of Symmetric, Diffuse Type (HS-D).—Four patients had such records and the electroencephalograms were taken at intervals of from one to six days after the onset of symptoms. All

4 patients were stuporous at the time. For 3 the clinical diagnosis was cerebral arteriosclerosis and thrombosis and for the fourth cerebral embolism. In addition to stupor, 2 patients showed hemiparesis. For 1 patient a repeat record taken six days later, when the patient was mentally alert, yielded a low degree of electroencephalographic abnormality of asymmetric, diffuse type.

Cerebrovascular Disease with High Degree of Electroencephalographic Abnormality, of Asymmetric, Focal Type (HA-F).—Two patients had such records. For 1 patient the electroencephalogram was recorded two days and for the other ten days after the onset of symptoms. Neither had any clouding of consciousness at the time. One patient, with a clinical diagnosis of polycythemia with cerebral thrombosis, presented hemiparesis with aphasia, and a repeat record taken three weeks later failed to show any change from the previous electroencephalogram. The other patient, with a diagnosis of cerebral thrombosis, had jacksonian seziures in addition to hemiparesis with aphasia, and a repeat record taken one year later failed to show any change in the electroencephalogram. For both patients the electroencephalographic focus corresponded to the anatomic focus postulated on the clinical evidence.

Cerebrovascular Disease with a High Degree of Electroencephalographic Abnormality of Asymmetric, Diffuse Type (HA-D).—Two patients had records of this type. For both the electroencephalograms were taken three days after the onset of symptoms. For both the clinical diagnosis was cerebral thrombosis; both presented the clinical signs of hemiparesis with stupor, and in both the side of greater electroencephalographic abnormality corresponded to the site of the focus as diagnosed from the clinical signs.

Cerebrovascular Disease with a High Degree of Electroencephalographic Abnormality of Parasymmetric, Focal Type (HP-F).—Two patients had records of this type, and both the electroencephalograms were recorded four weeks after the onset of symptoms. The clinical diagnosis for both was cerebral thrombosis; the clinical signs were hemiparesis with stupor, and the site of focal accentuation in the electroencephalogram corresponded in both instances to the focal lesion as diagnosed on the basis of the clinical signs.

Cerebrovascular Disease with a High Degree of Electroencephalographic Abnormality of Parasymmetric, Diffuse Type (HP-D).—Six patients had records of this type. The electroencephalograms were recorded at intervals of three days to eight months after the onset of symptoms. Four of the 6 patients were in stupor at the time of the electroencephalographic recording. Of these 4 patients, 3 died soon after admission to the hospital, 2 with a condition diagnosed as cerebral hemorrhage and the third with cerebral thrombosis. For the fourth

patient in stupor and for the remaining 2 patients who were not mentally clouded, the diagnosis of cerebral thrombosis was made. The clinical signs included hemiparesis with aphasia (1 patient), hemiparesis with stupor (2 patients), hemiparesis with memory defect (1 patient) and stupor without focal signs (2 patients). A repeat record taken two weeks later for the patient presenting hemiparesis with aphasia yielded a low degree of electroencephalographic abnormality of symmetric, diffuse type.

COMMENT

The great majority of patients (67 out of 95, or about 70.5 per cent) with cerebrovascular disease showed no delta activity in the electroencephalogram. This does not mean that all the records were normal, for in a previous survey,[3] of a series of 52 patients with cerebrovascular disease without delta activity, the records of 8 (approximately 15.5 per cent) showed an abnormally high degree of bilateral difference in the amount of alpha activity. In the present study, no corresponding measurements of alpha activity were made in the records used. However, if it is assumed that the percentage of patients with an abnormal bilateral difference in alpha activity is the same as that found in the earlier material, it may be expected that 55 per cent of the patients with cerebrovascular disease would yield an entirely normal electroencephalogram; 15.5 per cent, abnormal asymmetry of alpha activity, and the remaining 29.5 per cent, various types of records with delta activity, as recorded in table 1.

A positive correlation between symptoms and signs, on the one hand, and the electroencephalogram, on the other, was found to exist in only two respects: 1. In all the patients with an electroencephalographic focus, such a focus corresponded to the anatomic site of the lesion as diagnosed on the basis of the clinical facts. 2. All the patients with clouding of consciousness showed delta activity in the electroencephalogram. The latter correlation is hardly surprising, since previous investigators[4] made similar observations correlating delta activity with clouding of consciousness irrespective of its specific cause. All our patients with a high degree of electroencephalographic abnormality of symmetric, diffuse type (HS-D); asymmetric, diffuse type (HA-D), or parasymmetric, focal type (HP-F) showed clinically a disturbance in the state of consciousness, whereas only some of the patients with

3. Strauss, H.; Liberson, W. T., and Meltzer, T.: Electroencephalographic Studies: Bilateral Differences in Alpha Activity in Cases With and Without Cerebral Pathology, J. Mt. Sinai Hosp. **9**:957-962, 1943.

4. Gibbs, F. A.; Davis, H., and Lennox, W. G.: The Electroencephalogram in Epilepsy and in Conditions of Impaired Consciousness, Arch. Neurol. & Psychiat. **34**: 1133-1148 (Dec.) 1935. Strauss, H.: Clinical and Electroencephalographic Studies: Correlations of Mental, Electroencephalographic and Anatomic Changes in Cases with Organic Brain Disease, Am. J. Psychiat. **101**:42-50, 1944.

TABLE 1.—*Types of Electroencephalographic Abnormality and Clinical Findings in 95 Cases of Cerebrovascular Disease*

Type of Electroencephalogram *	No. of Cases	Age, Yr.	Diagnosis	Symptoms	No. of Cases	Clouding of Consciousness	Duration of Illness	Convulsions
No delta activity	67	25-77	Cerebral arteriosclerosis and cerebral thrombosis (66 cases); cerebral embolism (1 case)	Hemiparesis	24	—	1 day to 1 year	Grand mal (9 cases)
				Hemiparesis with aphasia	8	—	1 week to 4 mo.	Jacksonian seizures (4 cases)
				Hemiparesis with hemisensory defects	3	—	4 days to 3 weeks	
				Hemianopsia	6	—	2 weeks to 1 year	
				Hemianopsia with aphasia	1	—	2 months	
				Aphasia	2	—	5 weeks to 6 mo.	
				Headache and dizziness	4	—	3 mo. to 1 year	
				Psychosis	3	—	2 weeks to 9 mo.	
				Personality changes and memory defects	5	—	2 mo. to 1 year	
				Brain stem syndrome including 6 cases of posterior inferior cerebellar artery syndrome	11	—	2 days to 11 mo.	
LS-D †	8	32-70	Cerebral arteriosclerosis and cerebral thrombosis (7 cases); cerebral embolism (1 case)	Hemiparesis	1	—	6 months	
				Hemiparesis with aphasia	1	—	3 months	
				Headaches and dizziness	3	—	3 days to 1 year	
				Personality changes and memory defect	1	—	5 days	
				Drowsiness	1	+	5 days	
				Drowsiness with brain stem syndrome	1	+	1 day ‡	
LA-F	3	52-59	Cerebral thrombosis (3 cases)	Hemiparesis with aphasia	2	—	1 mo. to 1 year	
				Hemiparesis with hemisensory defect	1	—	8 days	
LP-F	1	32	Cerebral embolism (1 case)	Hemiparesis with aphasia	1	—	1 day ‖	
HS-D §	4	40-66	Cerebral arteriosclerosis and cerebral thrombosis (5 cases); cerebral embolism (1 case)	Stupor	2	+	1 day to 3 days	
				Stupor with hemiparesis	2	+	1 day to 6 days	
HA-F	2	36-66	Cerebral thrombosis (1 case); polycythemia (1 case)	Hemiparesis with aphasia	2	—	2 days# to 10 weeks**	Jacksonian seizures (1 case)
HA-D	2	48-68	Cerebral thrombosis (2 cases)	Stupor with hemiparesis	2	+	3 days	
HP-F ††	2	49-62	Cerebral thrombosis (2 cases)	Stupor with hemiparesis	2	+	4 weeks	
HP-D ‡‡	6	50-70	Cerebral thrombosis (4 cases); cerebral hemorrhage (2 cases)	Stupor	2	+	3 days to 8 mo.	
				Stupor with hemiparesis	2	—	3 days to 6 days	
				Hemiparesis with memory defect	1	—	3 weeks	
				Hemiparesis with aphasia	1	—	17 days §§	

* H indicates a high degree, and L a low degree, of electroencephalographic abnormality; S, A and P designate symmetric, asymmetric and parasymmetric distribution of delta activity, and F and D, focal and diffuse abnormality in the record.

† In 4 cases there were bursts of delta activity.

‡ A repeat record taken on the fourth day of illness, when the patient was not mentally clouded, showed a lessening in the degree of abnormality.

‖ A repeat record taken three days later showed a lessening in the degree of abnormality.

§ In 2 cases there were bursts of delta activity.

A repeat record taken three weeks later showed no change in the electroencephalogram.

¶ A repeat record taken in 1 case on the ninth day of illness, at which time the patient was not mentally clouded, yielded a low degree of electroencephalographic abnormality of asymmetric, diffuse (LA-D) type with bursts of delta activity.

** A repeat record taken one year later showed no change in the electroencephalogram.

†† In 1 case bursts of delta activity occurred.

‡‡ In 4 cases, in 3 of which the patients were stuporous at the time of the recording, bursts of delta activity were present.

§§ A repeat record taken two weeks later showed a low degree of electroencephalographic abnormality of asymmetric, diffuse (LA-D) type.

a low degree of electroencephalographic abnormality of symmetric, diffuse type (LS-D) or a high degree of abnormality of parasymmetric, diffuse type (HP-D) showed a similar clinical disturbance.

Normal records were seen for patients with hemispheric lesions producing hemiparesis, aphasia, hemianopsia and combinations of such symptoms, even though the lesion was of recent origin, e.g., for patients with hemiparesis as early as one day after the onset of symptoms. In other patients similar clinical findings were associated with abnormal electroencephalograms as late as one year after the onset of symptoms. These observations show that there is no correlation between the electroencephalogram, on the one hand, and the time interval and symptoms on the other, even though the two latter factors are considered in conjunction. However, for 4 of the 95 patients repeat records showed a diminution in the degree of abnormality as compared with the records taken earlier in the course of the disease.

In our series of 95 cases of cerebrovascular disease, epileptic attacks occurred in 14. In 9 cases they were generalized grand mal seizures, and in 5 jacksonian in character. Of the 5 cases of jacksonian seizures, only 1 yielded a high degree of electroencephalographic abnormality of asymmetric, focal type. None of the records showed a pattern indicative of a convulsive disorder, whereas such a pattern was found in 89 per cent of all cases of idiopathic epilepsy and in 26.5 per cent of our total material of cases with symptomatic epilepsy accompanying organic cerebral disease.

To ascertain electroencephalographic criteria that would aid in the differential diagnosis of tumor of the brain and cerebrovascular disease, results obtained in 95 cases of cerebrovascular disease were compared with those obtained in 162 verified cases of tumors of the cerebral hemispheres. Records with an electroencephalographic focus were obtained in 8 (8.5 per cent) of the 95 cases of cerebrovascular disease and in 119 (73 per cent) of the 162 cases of cerebral tumor. In the differential diagnosis, therefore, of a cerebrovascular accident and a cerebral tumor, a focally abnormal electroencephalogram favored the diagnosis of tumor in the ratio of 8:1. In cases of gliogenous and metastatic tumors of the brain, focal records were found in 77 per cent or about nine times as frequently as with cerebrovascular disease. In cases of meningioma focal records were found in 66 per cent, or a little less than eight times as frequently as with cerebrovascular disease.

Records of asymmetric abnormality were obtained in 7 (7.4 per cent) of the 95 cases of cerebrovascular disease and in 110 (68 per cent) of the 162 cases of hemispheric tumors. An asymmetric record, therefore, favored the diagnosis of tumor rather than that of cerebrovascular disease in the ratio of 9:1. Of 115 cases of gliogenous and metastatic tumors of the brain, an asymmetric record occurred in 85 (74 per cent),

A repeat record taken three weeks later showed no change in the electroencephalogram.
¶ A repeat record taken in 1 case on the ninth day of illness, at which time the patient was not mentally clouded, yielded a low degree of electroencephalographic abnormality of asymmetric, diffuse (LA-D) type with bursts of delta activity.
** A repeat record taken one year later showed no change in the electroencephalogram.
†† In 1 case bursts of delta activity occurred.
‡‡ In 4 cases, in 3 of which the patients were stuporous at the time of the recording, bursts of delta activity were present.

or ten times as frequently as in cases of cerebrovascular disease. Of 47 cases of meningioma, an asymmetric record occurred in 25 (53 per cent), or seven times as frequently as in cases of cerebrovascular disease.

An asymmetric record with a focus occurred in 5 (5.3 per cent) of the 95 cases of cerebrovascular disease and in 103 (63 per cent) of 162 cases of tumors of the cerebral hemispheres. In the differential diagnosis, therefore, of a cerebrovascular accident and a tumor of the cerebral hemispheres, an asymmetric record with a focus in the electroencephalogram favors the diagnosis of tumor in the ratio of 12:1. Of 115 gliogenous and metastatic brain tumors 79 (69 per cent) yielded an asymmetric record with a focus which is thirteen times as frequent as in cases of cerebrovascular disease. Of 47 meningiomas 24 (51 per cent) yielded an asymmetric record with a focus which is ten times as frequent as in cases of cerebrovascular disease.

TABLE 2.—*Incidence of Types of Records Associated with Cerebrovascular Disease and Hemispheric Tumors* *

Type of Record	Cerebrovascular Disease	All Hemisphere Tumors	Gliogenous and Metastatic Tumors	Meningiomas
Focal abnormality	8.5	73 (8:1)	77 (9:1)	66 (8:1)
Asymmetric	7.4	68 (9:1)	74 (10:1)	53 (7:1)
Asymmetric with focal abnormality	5.3	63 (12:1)	69 (13:1)	51 (10:1)
No delta activity	70.5	12 (1:6)	7 (1:10)	21 (1:3.5)

* Numbers indicate percentages of type of records in cases of cerebrovascular disease and hemispheric tumor. Numbers in parentheses indicate the approximate ratios of the occurrence of various types of records for the two conditions.

Records with no delta activity were obtained in 70.5 per cent of the cases of cerebrovascular disease and in only 12 per cent of the cases of hemispheric tumors (ratio 6:1). Normal records were obtained in 7 per cent of the cases of gliogenous and metastatic brain tumors, which is approximately one-tenth as frequent as in cases of cerebrovascular disease. Normal records were obtained in 21 per cent of the cases of meningiomatous tumors, a ratio of 1:3.5 as compared with cerebrovascular disease.

A comparison of the frequency of various types of electroencephalographic records in cases of cerebrovascular disease with the frequency of the same types of records in cases of hemispheric tumors as recorded in table 2, demonstrates the fact that certain types of records, i.e., asymmetric records and records with a focus, make the diagnosis of a brain tumor much more probable than that of a cerebrovascular lesion. It also shows that no one type of record is specific for one or the other

type of cerebral lesion. In addition to the types of records discussed, there are others, as noted in table 1, which cannot be used in the differential diagnosis of hemispheric tumor and cerebrovascular disease, since they occur with similar frequency in the two types of cerebral lesion.

SUMMARY

An attempt is made to correlate clinical signs, symptoms and duration of illness with the type of electroencephalogram in 95 cases of cerebrovascular accident.

Some correlation was found to exist between the state of consciousness and the type of electroencephalographic abnormality obtained.

No correlation was found to exist between the presence or absence of gross hemispheric lesions and the electroencephalogram other than that an electroencephalographic focus, when present, corresponded to the anatomic site of the lesion as diagnosed from the clinical findings.

In none of the 14 cases in which convulsive seizures, generalized or jacksonian, occurred did an electroencephalographic pattern indicative of convulsive tendency appear.

Whereas no type of electroencephalographic abnormality is found to be associated only with brain tumor or with cerebrovascular disease, there are certain types of electroencephalograms, i.e., records with asymmetry of delta activity and with a focus of abnormal activity, which are seen much more frequently with tumors than with cerebrovascular disease. Also, irrespective of the type of electroencephalographic abnormality, a diminution in the degree of abnormality in repeat records favors the diagnosis of cerebrovascular disease rather than that of cerebral tumor.

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Case Reports

OLIGODENDROGLIOMA OF THE CEREBELLUM

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AFTER the original histologic description of oligodendroglioma by Bailey and Cushing [1] in 1926, and later by Bailey and Bucy,[2] in 1929, it was generally believed that this tumor was a solitary benign lesion limited to one hemisphere. Dickson's report [3] of an oligodendroglioma of the third ventricle and the ventricular tumor reported by Thomas and Jumentié [4] aroused little suspicion of the varied nature of this tumor until the report by Martin [5] in 1931. This author divided oligodendrogliomas into two types: the midline type, which appears before the age of 25, produces signs of increased intracranial pressure early and hence has insufficient time to calcify; and the lateral, or hemispheric, type, which occurs at a later age, is slow growing and is usually calcified.

Löwenberg and Waggoner [6] found Martin's classification too limited and inadequate. In a study of 21 cases, they were able to divide the tumors into four types: (1) a bilateral type, involving both hemispheres; (2) a unilateral type, involving one hemisphere; (3) a ventricular type, and (4) oligodendroglioma of the basal nuclei.

In reviewing the literature, they noted other sites of involvement in the spinal cord, brain stem and cerebellum. The authors pointed out that the oligodendrogliomas frequently produced numerous nodules interconnected by streams of tumor cells. There was, likewise, a tendency of the cells to invade the meninges. This feature was also reported by Östenfeld,[7] by Martin [5] and, more recently, by Blumenfeld and Gardner.[8] The tendency to invade the meninges was likened to the

1. Bailey, P., and Cushing, H.: A Classification of the Glioma Group, Philadelphia, J. B. Lippincott Company, 1926.

2. Bailey, P., and Bucy, P. C.: Oligodendrogliomas of the Brain, J. Path. & Bact. **32**:735, 1929.

3. Dickson, W. E. C.: Oligodendroglioma of Floor of Third Ventricle, Brain **49**:578, 1926.

4. Thomas, A., and Jumentié, T.: Un cas de tumeur du ventricule lateral, Rev. neurol. **2**:202, 1928.

5. Martin, J. P.: Two Cases of Oligodendroglioma, with Remarks on the General Clinical Features of Such Cases, Brain **54**:330, 1931.

6. Löwenberg, K., and Waggoner, R. W.: Gross Pathology of the Oligodendrogliomas, Arch. Neurol. & Psychiat. **42**:842 (Nov.) 1939.

7. Ostenfeld, T.: Oligodendrocytoma nerv. opticorum hos et barn med mb. Recklinghausen, Hospitalstid. (Dansk. neurol. Selsk. Forh.) **79**:18, 1936.

8. Blumenfeld, C. M., and Gardner, W. J.: Disseminated Oligodendroglioma, Arch. Neurol. & Psychiat. **54**:274 (Oct.) 1945.

invasive character of a medulloblastoma. Bailey expressed the opinion that these tumors were related to the medulloblastomas. He stated:

> It is not surprising that these tumors should be related to the medulloblastomas for the oligodendroglia along the fibre tracts of the brain develops from the medulloblasts which wander into this region and there differentiate into either oligodendroglia or neuroglia.

Of 5 cases reported by Greenfield and Robertson[9] 3 were cystic tumors of the cerebral hemispheres. An oligodendroglioma growing in the wall of a cyst may alter the histologic character of the tumor. In these circumstances the cells assume a myxomatous appearance, with the production of a mucinoid exudate. A cystic tumor of the pons occurring in a 6 year old child was described by Hughes and Couper.[10] An oligodendroglioma of the cerebellum was reported by Garcavy.[11] A second case is reported here. The tumor was cystic.

REPORT OF CASE

History.—J. R., a girl aged 13, was admitted to the neurosurgical service at Temple University Hospital on April 27, 1945, because of constant headache, dizziness and staggering gait, with falling to the left side. Dizziness and staggering had appeared insidiously six weeks prior to her admission. About a week later the mother noted that the patient dragged her left leg and had some weakness of the left arm. Constant supraorbital headaches occurred four weeks prior to her admission. Walking became progressively more difficult, and she had fallen on several occasions. Falling was invariably to the left. Vomiting was present on one occasion.

Physical Examination.—The temperature was 99.2 F., the pulse rate 108 and the respiratory rate 10 to 18, per minute. The blood pressure was 100 systolic and 70 diastolic. General physical examination revealed nothing of significance. The patient was unusually bright and alert. The gait was widened and ataxic, with falling to the left. The head was held stiffly and inclined toward the left shoulder. The right pupil was larger than the left. The reactions to light and in accommodation were sluggish. A horizontal nystagmus of second degree was present on looking toward the left. Acute papilledema of 2 D. was present in each eye. The cranial nerves were otherwise normal. Speech and laughter were explosive. The deep reflexes were subdued. The left knee jerk was pendulous. Dysmetria was noted in the left upper and the left lower extremity in the finger to nose and heel to knee tests, respectively. Past pointing and hypotonus were pronounced on the left side. Impairment of fine associated movements and adiadokokinesis were observed on the left side. When the arms were held in the outstretched position, the left extremity drifted downward and outward. A Gordon-Holmes sign was positive on the left side.

Laboratory Studies.—The blood and urine were normal. Roentgenograms of the skull and chest were reported to show nothing abnormal.

Diagnosis.—The diagnosis was tumor of the left cerebellar hemisphere.

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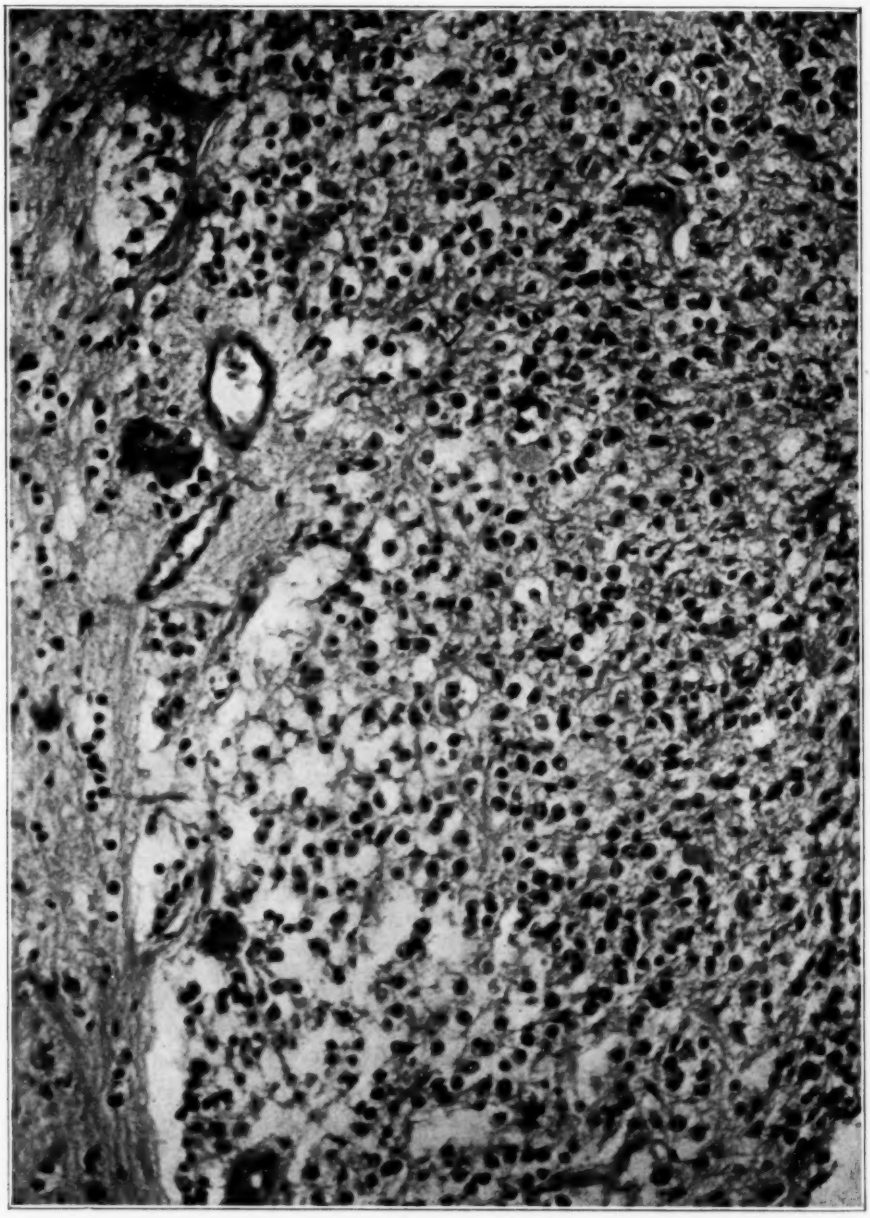
9. Greenfield, J. G., and Robertson, E. G.: Cystic Oligodendrogliomas of the Cerebral Hemispheres and Ventricular Oligodendrogliomas, Brain **56**:247, 1933.

10. Hughes, R. R., and Couper, E. C. R.: Oligodendroglioma in an Infant of Eight Months, Arch. Dis. Childhood **17**:147, 1942.

11. Garcavy, G.: Neuropathologia **5**:1778, 1936.

Operation (April 30, 1945).—Suboccipital craniectomy was performed with basal anesthesia induced with tribromoethanol U.S.P., followed by "pentothal" sodium anesthesia. With the patient in the face-down position, the usual horseshoe-shaped



Photomicrograph showing characteristic cellular structure of an oligodendroglioma.

incision was made. The right lateral ventricle was tapped, and clear ventricular fluid, under tremendous pressure, was slowly removed. The musculoaponeurotic flap was elevated, and the occipital bone over both cerebellar hemispheres was

removed by rongeur. The lamina of the atlas was exposed and removed. The dura was opened, and the left cerebellar hemisphere was observed to be more prominent than the right. The left cerebellar hemisphere was tapped, and at a distance of 2.5 cm. beneath the surface a large cystic tumor was encountered. The cortex was incised and the wall of the cyst isolated with pledgets of cotton. The cystic tumor was eventually teased out of its bed and entirely removed. The tumor was approximately the size of a hen's egg. The dura was allowed to remain open and the musculoaponeurotic flap replaced and carefully sutured with double tiers of silk. One drain was placed in the epidural space.

Microscopic Diagnosis.—The histologic diagnosis was oligodendroglioma (figure).

Follow-Up Report.—The patient made an uneventful recovery and the papilledema and cerebellar symptoms rapidly subsided. At the time of writing, two years later, the patient is entirely free from symptoms except for mild residual cerebellar signs on the left side and persistent horizontal nystagmus of second degree on left lateral gaze. She attends school daily and does well in typing despite her mild cerebellar deficiency.

CONCLUSION

The symptoms and gross appearance of the tumor at operation would lead one to believe that it was a classic astrocytoma. Careful studies of several histologic sections by several pathologists indicated without doubt that the tumor was an oligodendroglioma. The case presented an unusual manifestation of the oligodendroglioma. This report, as well as those of Martin,[5] Greenfield and Robertson,[9] Hughes and Couper,[10] Blumenfeld and Gardner[8] and Löwenberg and Waggoner,[6] indicates that the oligodendroglioma may assume a variety of pathologic forms.

EPENDYMOMA IN THIRD AND FOURTH VENTRICLES WITH IMPLANTS IN SPINAL SUBARACHNOID SPACES

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IT HAS generally been felt that ependymomas arising within the brain do not spread or form implantations on distal parts of the cerebrospinal axis. However, a few cases have been reported in which it must be concluded that such spread or implantation did occur. The first such report of such a case is believed to be that of Spiller,[1] who later referred to a similar case.[2] Thereafter, Cairns and Russell[3] reported the case of an ependymoma of the roof of the fourth ventricle with small metastases to the pia mater of the fifth thoracic segment. Tarlov[4] described the observation at operation of a pea-sized cervical implantation, and Dyke and Davidoff[5] referred to 3 additional cases in which an ependymoma was associated with spinal implants. Two cases were added to this series by Tarlov and Davidoff.[6] In a recent study of gliomas producing "meningeal gliomatosis," Polmeteer and Kernohan[7] mentioned 3 cases of ependymoma of ventricular origin which was associated with leptomeningeal involvement.

REPORT OF A CASE

History.—A white woman aged 46 was admitted to the Illinois Neuropsychiatric Institute on Aug. 7, 1944 as a transfer patient from another hospital. She stated that for the preceding seven years she had experienced severe periodic pain in the lower part of the back, lasting two to three days and radiating down the right leg to the calf. Straining, working or coughing made the pain

From the Department of Neurology and Neurological Surgery, Illinois Neuropsychiatric Institute, University of Illinois College of Medicine, Chicago.

1. Spiller, W. G., and Hendrickson, W. F.: A Report of Two Cases of Multiple Sarcomatosis of the Central Nervous System and of One Case of Intramedullary Sarcoma of the Spinal Cord, Am. J. M. Sc. **126**:10-33, 1903.

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worse, while rest in bed produced some relief. In February 1943 she became aware of failure in vision, for she found it difficult to do fine work or to read small print. Partial relief was obtained with the use of glasses, but her vision became progressively worse. Several weeks prior to admission to the hospital she began to feel weakness and numbness in the right lower extremity, which progressed until she remained in bed a large part of the time. She had no urinary symptoms.

Roentgenologic Examination.—Roentgenologic studies, made at the first hospital, had revealed some enlargement of the sella turcica, with prominent destruction of the posterior clinoid processes. Spinal puncture had revealed a bloody fluid under a pressure of 400 mm. of water, and on Aug. 4, 1944 a ventriculographic

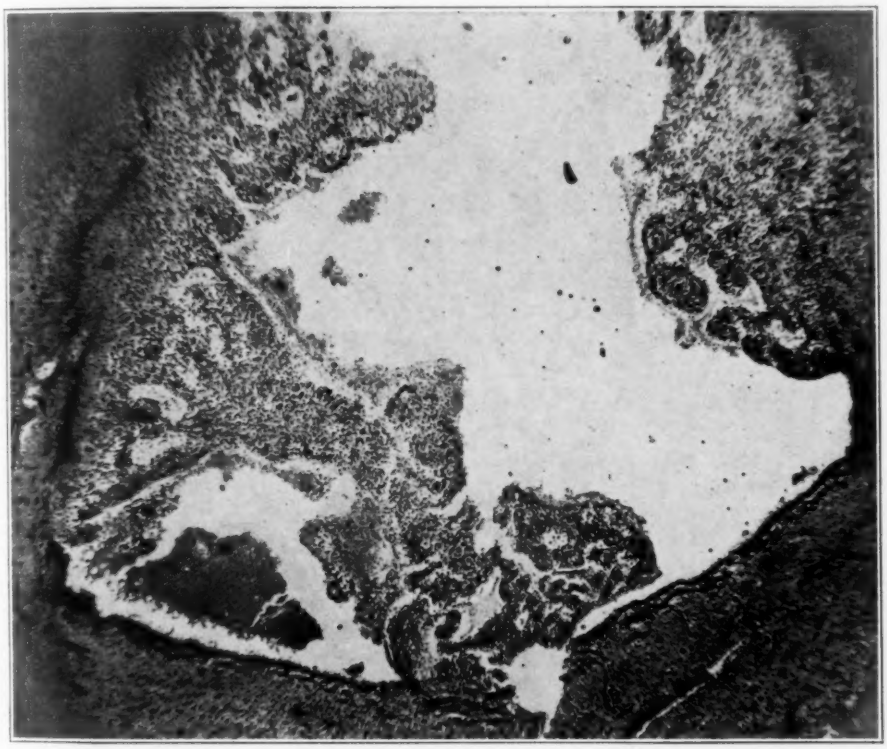

Fig. 1.—Infundibulum of the third ventricle. The tumor is proliferating into the cavity from the ependyma. Hematoxylin and eosin stain.

study was made through a burr hole in the left parietal region. The roentgenograms showed moderate dilatation of the lateral ventricles, with a small amount of air in the posterior part of the third ventricle and no filling of the fourth ventricle. No shift of the ventricles was noted. After the ventriculographic examination polyuria developed.

Examination.—On her admission to the Illinois Neuropsychiatric Institute the abnormal physical findings included bilateral mild papilledema, severe impairment of visual acuity, moderate weakness and atrophy of the right lower extremity with absence of the tendon reflexes, and absence of the abdominal reflexes. No sensory loss was demonstrated.

Operation.—At operation, on August 9, the dilated right lateral ventricle was entered through a small bone flap in the right frontal region, and the foramen of Monro was exposed. A lighted retractor was inserted through the foramen of Monro into the third ventricle, and an inoperable lesion of the floor of the third ventricle was visualized. Severe hyperthermia developed after operation, and the patient died within twenty-four hours.

Autopsy.—Autopsy revealed acute hemorrhagic cystitis and pyelitis, bilateral hydronephrosis with hydroureters, leiomyomas of the uterus and minimal congestion of the liver and lungs. The spinal cord was removed within the dural sac. It was noted that the lower end of the spinal canal was enlarged and that the sacral

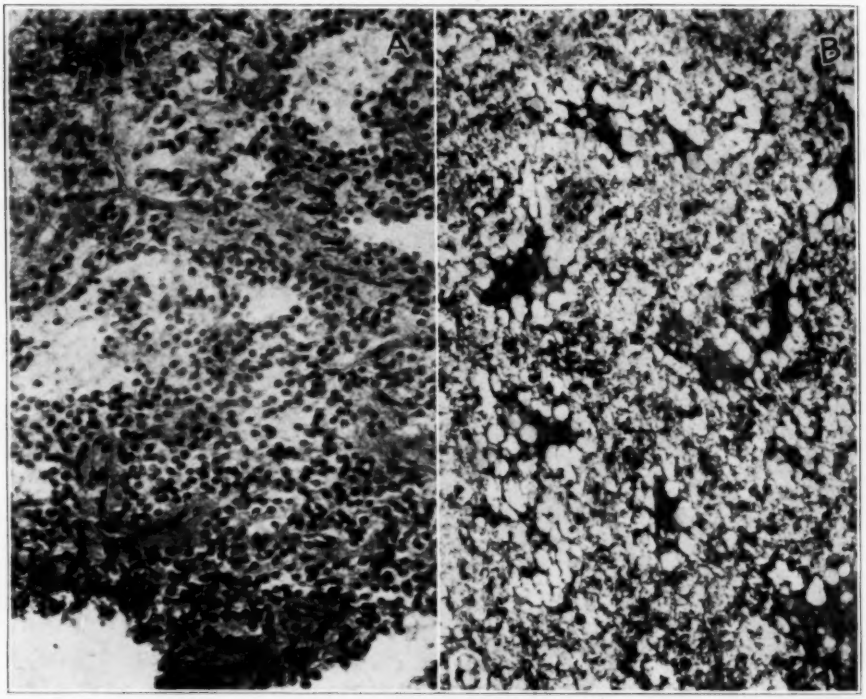

Fig. 2.—Tumor of the third ventricle. *A*, hematoxylin and eosin stain; *B*, thionine stain.

extremity of the dural sac disappeared into soft tissue, which appeared to be neoplastic since it was redder and more necrotic than the normal epidural tissue in the thoracolumbar region.

The contour of the brain appeared normal except for the area of trephination in the left parieto-occipital region and the operative laceration of the right frontal lobe. No herniation from pressure was evident, and the cerebral convolutions were not flattened. The right lateral ventricle contained a blood clot of considerable size, which filled the posterior part of the ventricle and extended into the third ventricle. No evidence of tumor was present in the lateral ventricles; the choroid plexuses were normal, and the ependymal lining was smooth. A small amount of grayish, translucent material was evident about the floor of the third and fourth ventricles. No evidence of infiltration of the leptomeninx in the basilar cistern or around the brain stem could be seen. The leptomeninges over the spinal cord

appeared thickened and brownish, as though from old hemorrhage. In the thoracic region, thickening of the leptomeninx became more obvious, and there was a mass on the posterior aspect of the lumbar portion of the spinal cord about 1.5 cm. in diameter and 3 mm. in thickness. This tumor extended down among the roots of the cauda equina to the lower end of the dural sac, and apparently beyond into the subjacent epidural space.

The tissue was embedded in pyroxylin, since the tumor was fragile and fell to pieces in frozen sections. The sections were stained with hematoxylin and eosin, thionine and the Van Gieson method.

In a block through the infundibulum of the third ventricle, the tumor could be seen (fig. 1) to be proliferating from the ependyma. There was no tendency to

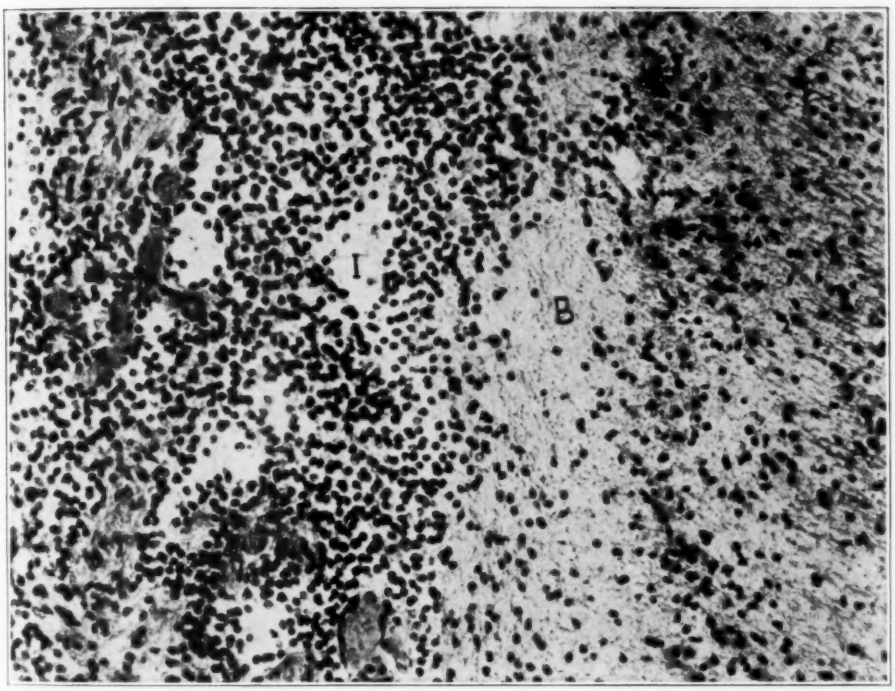

Fig. 3.—Tumor from the fourth ventricle. Note the sharp margin against the bulb. *T* indicates the tumor; *B*, the bulb. Van Gieson stain.

invade the brain tissue; the tumor grew as a loose mass of cells into the cavity of the ventricle. The tumor was clearly delimited by a dense layer of cells, which at places formed small ependymal canals.

Because of its poor vascularization, extensive degenerative cavities were scattered throughout; these were filled with a rather homogeneous material, which was almost unstained with eosin or fuchsin but stained reddish with thionine (fig. 2 *B*). In relatively healthy parts of the tumor the neoplastic cells could be seen to be polygonal, with considerable cytoplasm and definite cell boundaries. They had a tendency to arrange themselves about the blood vessels (fig. 2 *A*). In most parts of the tumor it was difficult to distinguish cellular boundaries; the fixation had produced a sort of syncytium, in the meshes of which the oval nuclei were embedded. Innumerable vacuoles were produced by this shrinkage (fig. 2 *B*).

The absence of macrophages would lead one to suppose that these vacuoles did not contain fat, but this I was unable to prove.

The tumor in the fourth ventricle was in all respects identical with that in the third. It formed a thick layer over the ventricular surface and was sharply circumscribed against the underlying bulb (fig. 3).

The implant in the subarachnoid space varied somewhat in structure (fig. 4 *A*). The cells were often more elongated and mingled with connective tissue. But the same degenerative process was prevalent, and the tumor was sharply delimited by the pia mater.

About the roots of the cauda equina the syncytial appearance was again more evident and the nuclei were more scattered. Small blood vessels with proliferated

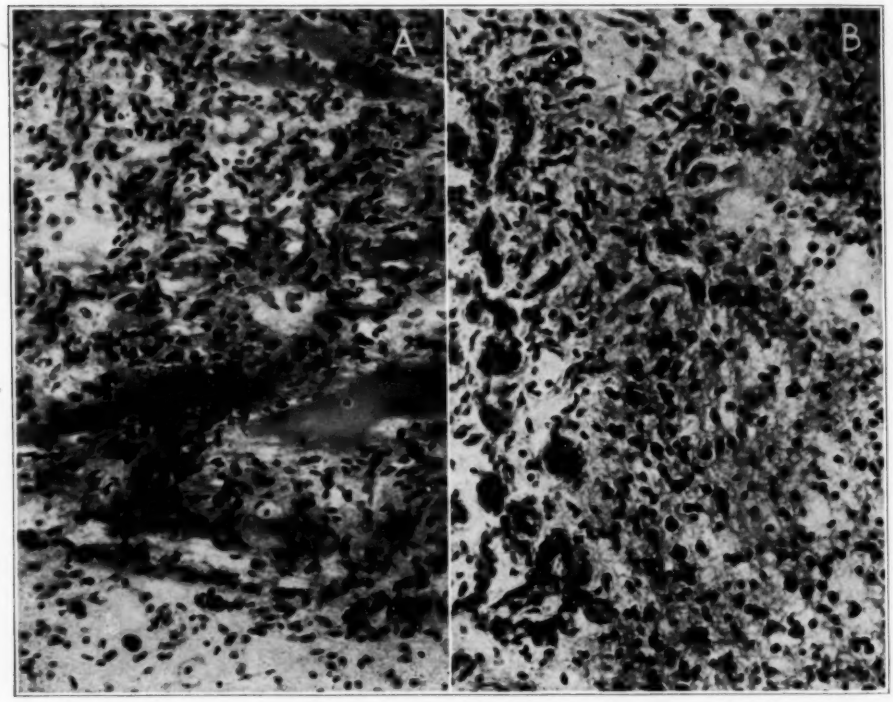

Fig. 4.—Tumor implants. *A*, from the lumbar spinal subarachnoid space, with the spinal cord (*C*) below. *B*, from the region of the cauda equina. Thionine stain.

endothelium were numerous, and large degenerative cavities were present (fig. 4 *B*).

In spite of the somewhat different microscopic appearance, there is good reason to suppose, from the uniform manner of degeneration, that the tumor in the subarachnoid spaces, over the spinal cord and about the roots of the cauda equina arose by implantation from the ventricular tumor, although finer cytologic study was impossible.

Many roots of the cauda equina were normal, but others were extensively degenerated—the myelin sheaths had entirely disappeared, and the neurilemma and endoneurium were thickened. I was not fortunate enough to discover the point of exit of the growth from the dural sac, but infiltrating fingers of tumor tissue were seen in the dura mater and in the loose connective tissue external to it, sharply circumscribed as though in well defined spaces near the nerves and

agreeing in structure with the tumor within the sac. The mass in the epidural space below the dural sac was intimately intermingled with connective tissue but otherwise resembled the nodules in the cauda equina.

Since there is no reason to suppose that an independent gliomatous tumor developed in the lower epidural space, it may be supposed that implantation from the ventricular tumor utilized the same pathways that are so often demonstrated with iodized oil U.S.P. and other radiopaque oils after injection into the lumbar sac.

COMMENT

Analysis of the reports of subarachnoid implantations or seedings makes it probable that such metastatic tumors are not so uncommon as they were once considered to be. Symptomatic reference to the spinal cord in a case of ependymoma of the brain should raise the suspicion of the existence of such an implant. In view of the small size of implants that may occur, it is possible that the implant may remain relatively asymptomatic, so that careful examination of the spinal cord would be required to disclose its existence. In Cairns and Russell's case,[3] the spinal implants consisted of a relatively inconspicuous group of glistening, pinhead-sized nodules in the thoracic pia mater. Indeed, Dyke and Davidoff [5] advocated prophylactic radiation of the spinal axis in any case of verified ependymoma of the cerebrum or cerebellum, because they felt that the observation of spinal implants in 3 of their 8 cases of ependymoma indicated a relatively high incidence of spinal implants.

From the cases reported it may be inferred that spinal implantation is favored by the situation of the primary tumor in the third or fourth ventricle, where, presumably, the action of the force of gravity would tend to spread desquamations or metastases through the foramen of Magendie into the adjoining subarachnoid spaces. Cairns and Russell [3] expressed the belief that the potentiality of a glioma to disseminate metastases in the cerebrospinal pathway depends as much on the site of the tumor in relation to this pathway as on the intrinsic malignancy of its component cells. The possibility of seeding of this relatively benign glioma by operation, as mentioned by Tarlov and Davidoff,[6] must be remembered; but in most of the instances reported this was improbable, since the implants were symptomatic and well established at the time of the initial operation.

SUMMARY

A case is reported in which an ependymoma of the floor of the third and fourth ventricles had produced implants in the spinal subarachnoid spaces, one of which, in the sacral extremity of the dural sac, appeared to have spread into the epidural tissue, probably along the perineurial spaces.

At least 12 similar cases have been described previously, so that it is concluded that implantation or seeding in the spinal subarachnoid spaces is a distinct possibility in any given case of ependymoma of the brain.

The seeding of this usually benign type of tumor is probably favored by its common site in the ventricular system.

St. Vincent's Hospital.

News and Comment

THE AMERICAN BOARD OF NEUROLOGY AND PSYCHIATRY, INC.

The following candidates successfully passed the last examination of the American Board of Neurology and Psychiatry, held in Washington, D. C., in May, 1948:

Psychiatry.—Charles C. Adams, Murfreesboro, Tenn.; Edward R. Adelson, Brooklyn; Renato J. Almansi, New York; Calvin Layle Baker, Dayton, Ohio; Louis M. Berger, North Hollywood, Calif.; Otto Billig, Nashville, Tenn.; Joseph B. Bounds, North Little Rock, Ark.; C. H. Hardin Branch, Philadelphia; Henry H. Brewser, Boston; Martha Brunner-Orne, Boston; Howard M. Burkett, Ann Arbor, Mich.; Robert C. Burnham, Washington, D. C.; Ewald W. Busse, Denver; Louis Byer, Tuscaloosa, Ala.; Curtis R. Chaffin, El Reno, Okla.; Ewin Summers Chappell, Topeka, Kan.; Emanuel Chat, Canandaigua, N. Y.; Daniel Duncan Chiles, Radford, Va.; Irving Chipkin, New York; Lilli M. V. Cobliner, New York; R. Robert Cohen, Denver; George Morterud Cowan, Fort Snelling, Minn.; Jean G. N. Cushing, Baltimore; Mary Eyman McKinnis Cushing, Baltimore; Nicholas Peter Dallis, Toledo, Ohio; Daniel Dancik, Huntington Station, N. Y.; * Stanley M. Dillenberg, New York; Martin Dollin, Jackson Heights, N. Y.; Albert Etienne Dreyfus, Perry Point, Md.; Paul Lane Dunstan, North Grafton, Mass.; Abraham Edelstein, Northport, N. Y.; Jack R. Eidelman, St. Louis; David S. Evans, Brentwood, N. Y.; Joseph Arthur Farmer, Hartford, Conn.; Elizabeth D. Fletcher, Little Rock, Ark.; Harold Vandiver Ford, Topeka, Kan.; Bernard Frazin, Fort Harrison, Ind.; Lawrence M. Gahagan, New York; Howard P. Gilbert, Denver; Margaret C. L. Gildea, St. Louis; Ernest Stewart Goddard, London, Ontario, Canada; Daniel Lawrence Goldstein, New York; Charles E. Goshen, New York; * Eugene Willard Green, Staten Island, N. Y.; Marcus Guensberg, Kaneohe, Hawaii; Maurice Ernest Hermann, Northport, N. Y.; Irene L. Hitchman, Sykesville, Md.; Beryl Jaffe, Philadelphia; Edward R. Janjigian, Kingston, Pa.; Delbert Pinkerton Johnson, St. Joseph, Mo.; Dorothy Johnston, Danville, Pa.; Albert J. Kaplan, Philadelphia; Nathan S. Kline, Lyons, N. J.; Francis O. Lamb, Chicago; Henry P. Laughlin, Chevy Chase, Md.; Harry Luloff, New York; Frank Harper Luton, Nashville, Tenn.; John W. Lyons Jr., Havertown, Pa.; Arthur J. Macht, Milwaukee, Wis.; George J. Martin, Norristown, Pa.; Joseph H. Merin, New York; Harold L. Meyers, Topeka, Kan.; Benjamin F. Morton, Birmingham, Ala.; Henry J. Myers, Washington, D. C.; Hans Oppenheimer, Memphis, Tenn.; William L. Peltz, Philadelphia; A. Z. Pfeffer, New York; H. Tharp Posey, New Orleans; Thomas M. Proctor, Beacon, N. Y.; Gerald R. Rausch, Kirkwood, Mo.; William G. Reese, Baltimore; David Rest, Chicago; Waldemar G. Richter, Bedford, Mass.; Kurt E. Rose, Boston; Clifford J. Sager, New York; Leon Salzman, Washington, D. C.; Louis Schneidman, Van Nuys, Calif.; Louis M. Shapiro, Lyons, N. J.; Marshall F. Shields, Wallingford, Pa.; Walworth Slenger, Ann Arbor, Mich.; Sophie Schroeder Sloman, Chicago; Philip E. Stahl, Los Angeles; John Stathakis, North Little Rock, Ark.; Rutherford B. Stevens, Topeka, Kan.; Alexander Thomas, New York; Lieut. Comdr. S. V. Thompson (MC), U. S. N. R.; William Arnold Tice, New Orleans; Sarah Sheldon Tower, Baltimore; * Theodore J. C. von Storch, Albany, N. Y.; Nathaniel Warner, New York; Charles Watkins,

* The asterisk denotes complementary certification.

Durham, N. C.; Sidney U. Wenger, Philadelphia; William C. Wermuth, Philadelphia; Henry Wexler, Topeka, Kan.; Paul D. Williams, Indianapolis.

Neurology.—Ralph W. Barris, Grand Rapids, Mich.; Kendall B. Corbin, Rochester, Minn.; * Harold H. Dubner, Chicago; James Rowland Gay, Rochester, Minn.; Hannibal Hamlin, Providence, R. I.; Nathaniel Rogers Hollister, Dayton, Ohio; Waldemar Curtis Rasmussen, Rochester, Minn.; * Charles Russman, Middletown, Conn.; Mortimer Fromberg Shapiro, New York; Frederick M. Stark, Rochester, Minn.

Neurology and Psychiatry.—Allan Archibald Bailey, Rochester, Minn.

EXAMINATION FOR MEDICAL OFFICERS ANNOUNCED

The United States Civil Service Commission has announced an examination for filling medical officer (rotating intern and psychiatric resident) positions in St. Elizabeth's Hospital, Washington, D. C.

Medical officers (rotating interns) are paid $2,200 for the first year and $2,400 for the second year. Medical officers (psychiatric residents) are paid from $2,400 to $4,100 a year, depending on the amount of approved postgraduate training the applicant has completed. Appointments are open for July 1, 1949. Internships consist in two years of rotating service, and psychiatric residencies consist in one to three years in psychiatry. To qualify for internships, applicants must be third or fourth year students in an approved medical school; however, they may not enter on duty until they have successfully completed the full course of study. Applicants for psychiatric residencies must be graduates of an approved medical school, with the degree of Doctor of Medicine; in addition, they must have completed an approved internship or must now be serving such an internship. No written test is required for the medical officer positions. Details about the requirements are given in the examination announcement.

Interested persons may secure information and application forms from the United States Civil Service Commission, Washington 25, D. C., from most first class and second class post offices, and from Civil Service regional offices. Applications will be accepted until further notice in the Commission's Washington office.

THIRD NATIONAL CONFERENCE ON PSYCHODRAMA, SOCIODRAMA, SOCIOMETRY AND GROUP PSYCHOTHERAPY

The Third National Conference on Psychodrama, Sociodrama, Sociometry and Group Psychotherapy, sponsored by the Moreno Clinic and the Psychodramatic Institute, will be held in Beacon, N. Y., Sept. 4 to 6, 1948. The conference will cover psychodrama, sociodrama, sociometry and group psychotherapy. The theme of the conference will be "Training in Human Relations." The first day will be devoted to psychodrama; the second, to sociodrama, and the third, to sociometry and group psychotherapy.

Further information may be secured from the Moreno Clinic, Beacon, N. Y.

MEDICAL JOURNALS NEEDED ABROAD

Requests from libraries, medical societies and individual physicians in various devastated countries are being received almost daily. If physicians have extra copies of medical journals or copies for which they have no further use, it will be greatly appreciated if they will send them to the headquarters of the Association for distribution abroad.

SOUTHERN SOCIETY OF ELECTROENCEPHALOGRAPHY

The Southern Society of Electroencephalography was organized by the executive council of that society in Atlantic City, N. J., June 11, 1948. The first general meeting will be held at the time and place of the next meeting of the Southern Psychiatric Association. All persons in the southern area interested in becoming members of this organization are requested to communicate with the acting secretary-treasurer of the soceity, Dr. Samuel C. Little, 2111 Highland Avenue, Birmingham 5, Ala.

CORRECTION

In the article by Dr. Sydney Sunderland, entitled "Rate of Regeneration in Human Peripheral Nerves," published in the September 1947 issue (ARCH. NEUROL. & PSYCHIAT. **58**:251), an error occurred in table 5, page 262, referring to case 137; i. e., "2.0 above M. F. E." should read "20.0 above M. F. E."

Abstracts from Current Literature

EDITED BY DR. BERNARD J. ALPERS

Physiology and Biochemistry

STUDIES ON THE METABOLISM OF BRAIN SUSPENSIONS: IV. ANAEROBIC GLYCOLYSIS. K. A. C. ELLIOTT and M. HENRY, J. Biol. Chem. **163**:361, 1946.

Elliott and Henry studied the factors affecting anaerobic glycolysis of suspensions of cortex. They found that maximum stimulation with pyruvate is reached with a concentration of pyruvate of about 5×10^{-5} millimols. The rate is increased briefly by a preliminary period of aerobiosis and continuously by the presence of a trace of oxygen. These effects are shown to be due to the production, during respiration, of a concentration of pyruvate sufficient to stimulate the glycolysis. The rate is increased by washing the suspension with an isotonic medium. This effect is shown to be due to the removal of a system which destroys pyruvate.

The rate per unit weight of tissue is decreased by increasing concentration of tissue. This effect is not more than partly due to increase in pyruvate-destroying factors. Added lactate inhibits glycolysis somewhat in the absence, but not in the presence, of added pyruvate. Serum inhibits considerably. This effect is not due to the destruction of pyruvate. In Ringer-phosphate solution the production of lactic acid is considerably lower than in Ringer-bicarbonate solution, and a large amount of unidentified acid is produced. The activity of cortex slices is much more variable than that of suspensions. The rate of glycolysis for dilute suspensions of cortex is nearly the same as the average rate for slices.

PAGE, Cleveland.

STUDIES ON CHOLINE ACETYLASE: II. THE FORMATION OF ACETYLCHOLINE IN THE NERVE AXON. D. NACHMANSOHN, H. M. JOHN and M. BERMAN, J. Biol. Chem. **163**:475, 1946.

The high concentration of cholinesterase in the axon and its exclusive localization at the neuronal surface are among the essential facts supporting the concept that the release of acetylcholine is directly associated with the nerve action potential, i. e., with the conduction of the nerve impulse. The enzyme found in the axon is an esterase specific for acetylcholine. The presence of a specific and highly active enzymatic mechanism for the removal of acetylcholine in the axon indicates that the ester is metabolized there at a high rate and suggests the possibility that acetylcholine may be formed in the axon as well as at the synapse. As shown by Nachmansohn and Machado, acetylcholine is synthesized by an enzyme system, choline acetylase, which can be extracted from brain but not from other organs. Several properties of the enzyme have been described in previous communications. It appeared of interest to test whether or not choline acetylase occurs in the axon. Choline acetylase has been extracted from the sciatic nerve of the rabbit. A solution prepared from 1 Gm. of fresh nerve fibers forms 70 to 90 micrograms

of acetylcholine per hour, as compared with 150 to 200 micrograms per hour in solutions prepared from rat or guinea pig brains. The presence of the complex energy-requiring enzyme system in the nerve axon, i. e., that part of the neuron which does not contain nerve endings and cell bodies, is further evidence in support of the assumption that acetylcholine may be essential not only for the transmission of the nerve impulse across the synapse but also for its propagation along the axon. In the degenerating nerve fiber the enzyme activity decreases slowly. Forty-eight hours after section only 20 to 25 per cent of the initial activity is lost. At that time conductivity is still possible. Seventy-two hours after section, when conductivity has disappeared, the enzyme activity has decreased by two thirds. No enzyme was found six days after section. The results are consistent with the assumption that the release of acetylcholine is associated with conductivity.

PAGE, Cleveland.

ON RE-INNERVATION OF PARETIC MUSCLES BY THE USE OF THEIR RESIDUAL NERVE SUPPLY. H. E. BILLIG, A. VAN HARREVELD and C. A. G. WIERSMA, J. Neuropath. & Exper. Neurol. **5**:1 (Jan.) 1946.

The present study was undertaken in an effort to determine whether it was possible to improve the function of a paretic muscle by inducing increased branching in the intact motor fibers and reinnervating with these extra nerve branches muscle fibers which had lost their nerve supply. Studies were made on patients with poliomyelitis who had had the acute symptoms of the disease at least one year before, but in most instances considerably longer. The paresis present about one year after the acute attack could be regarded as a permanent disability. In the course of the study, motor nerves were interrupted by one of two methods: (1) the manual method, which consisted in forceful massage of the muscle or, later, contusion with a special hammer, and (2) the surgical method, which consisted in exposure of the nerve and subsequent crushing. Muscle power was rated according to a modification of the Lovett system, which recognizes six ratings, ranging from 0 to 6, the last designating normal power. Intermediate ratings used in this study ranged from zero, which is 1, to good, which is 12.

In selected cases muscle action potentials were recorded with a Matthews oscillograph. The result of the surgical treatment was a definite mean improvement in the muscles with a low initial rating. The muscles with a high initial rating showed a mean loss of muscle power. The application of the surgical method should remain restricted to muscles which have a low Lovett rating.

The manual treatment produced a similar mean improvement in muscle power, which was not restricted to the muscles of low initial rating. The manual treatment, thus, can be applied to all paretic muscles. Nontreated (control) muscles showed only the slightest changes in muscle power during the period of observation. It was observed that there was no distinct influence on the duration of the paresis; nor was the patient's age a factor. The effect of manual treatment reached its maximum in about one year. There is evidence that the surgically treated muscles continued to improve after that period. Repetition of the manual treatment after preceding surgical or manual treatment increased the rating of muscle power only slightly, if at all. Parallel increases in muscle power and increases in the action potentials were observed.

GUTTMAN, Philadelphia.

THE POSTERIOR TIBIAL REFLEX: A REFLEX OF SOME VALUE IN THE LOCALIZATION OF THE PROTRUDED INTERVERTEBRAL DISC IN THE LUMBAR REGION. R. M. PEARSON DONAGHY, J. Neurosurg. **3**:457 (Sept.) 1946.

Donaghy studied the posterior tibial reflex in 100 control subjects, in 5 patients with surgically verified ruptured disk and in 7 patients with myelographic evidence of ruptured intervertebral disk. All herniations occurred between the fourth and the fifth lumbar vertebra. In 31 per cent of the normal (control) subjects the posterior tibial reflex was absent bilaterally, and in 28 per cent it was elicited only by reenforcement. The reflex was absent in the 5 patients with verified protrusion of the disk and in 3 of the 8 patients with myelographic evidence of herniation. The reflex in these patients was absent on the affected side only.

Donaghy states that the posterior tibial reflex is of value in diagnosis of protruded intervertebral disk between the fourth and fifth lumbar vertebrae. To be significant, the reflex must be absent unilaterally.

TOZER, Philadelphia.

PSYCHOPHYSIOLOGICAL RELATIONS IN CASES WITH HEAD INJURIES. JURGEN RUESCH, Psychosom. Med. **7**:158 (May) 1945.

Ruesch compares the psychologic and physiologic functions of patients with post-traumatic syndromes with corresponding functions of patients with disease of the brain, on the one hand, and with psychoneuroses, on the other. The respiratory function under basal conditions, during work and during muscular tension, and the heart rate during endurance to pain and fatigue were correlated with the results of tests of intelligence and personality in five groups of patients. These groups comprised 16 patients with disease of the brain, 29 patients with recent head injuries, 29 patients with post-traumatic syndromes with signs of cerebral damage, 28 patients with post-traumatic syndromes without signs of cerebral damage and 30 patients with psychoneurosis. In addition, 15 normal controls were subjected to some of the tests.

Basal ventilation volume and tidal air increased in relation to rising neurotic scores obtained in the Minnesota Multiphasic Personality Inventory. The metabolic rate under basal conditions and during work was higher for patients with signs of damage to the brain than for patients with no evidence of a cerebral lesion.

Endurance of strain was determined by observing the change in heart rate following work of the hand during complete ischemia. All patients with head injury showed a smaller increase in heart rate than did the patients with cerebral disease or psychoneurosis or the normal controls. Return of the heart rate to the initial level was delayed in the patients with acute head injuries.

Cases of post-traumatic syndrome, without signs of cerebral damage, resembled the cases of psychoneurosis more than the cases of post-traumatic syndrome, with evidence of lesions of the brain. Patients with acute head injury were in many respects the most normal of all the patients studied.

The author concludes that there is an antagonism between the presence of damage to the brain and psychoneurosis. Patients with a post-traumatic syndrome usually showed the presence of both components, but with decided predominance of the neurotic aspects.

WERMUTH, Philadelphia.

MIGRAINE AND THE SYMPATHETIC NERVOUS PATHWAYS. G. F. ROWBOTHAM, Brit. M. J. **2**:319 (Sept. 7) 1946.

It is generally agreed that the pain of migraine is probably due either to excessive spasm or to dilatation of the arteries of the scalp and dura mater, but the reason for this excessive spasm or dilatation has remained conjectural. It has been suggested that a migrainous person is born with an unstable mechanism in the hypothalamus which reacts excessively to the multitudinous stimuli and causes explosive or dysrhythmic messages to be sent to the large blood vessels of the head. Rowbotham reasoned that if these disordered messages from the hypothalamus are transmitted by the motor autonomic pathways to the vessels concerned, section of those pathways should give relief. He tested this hypothesis on 4 patients with migraine, removing part or all of the upper cervical sympathetic ganglion, stripping the outer coats of the common internal and external carotid arteries and ligating and dividing the external carotid artery. He was able to obtain complete relief in 3 of the 4 patients for a period of several months to over a year after operation. The other patient stated his belief that the operation was of some help, but he still had headaches. Rowbotham attributes failure in the fourth case either to the possibility that the unstable mechanism lay in the vessels rather than in the hypothalamus or to removal of the whole rather than only the lower half of the cervical sympathetic ganglion.

On the assumption that the pain of migraine arises in the arteries of the head from excessive spasm or dilatation, that in some cases the pain is transmitted to the brain through the upper part of the posterior root of the trigeminal nerve and that the arterial spasm is aroused by dysrhythmic messages passing along the motor sympathetic pathways, Rowbotham has constructed a neural mechanism to account for many of the phenomena of migraine. ECHOLS, New Orleans.

REFLEXES ELICITED BY VISCERAL STIMULATION IN THE ACUTE SPINAL ANIMAL. C. B. B. DOWNMAN and B. A. MCSWINEY, J. Physiol. **150**:80, 1946.

Downman and McSwiney studied reflexes induced by stimulation of the intestine, mesentery and pancreas in decerebrate, decapitate and spinal cats. The reflexes comprised increases in blood pressure and movements of the skeletal muscles. Spinal preparations manifested both types of reflexes. Decapitate animals likewise responded, but less consistently and with weaker reflexes; movements of the skeletal muscles were not always present. Decerebrate animals responded to visceral stimulation with changes in blood pressure only. Stimulation of the mesentery or pancreas was more effective than stimulation of the intestine. Reflex responses to visceral stimuli were sometimes weak or absent when somatic responses to cutaneous stimuli were vigorous. THOMAS, Philadelphia.

Psychiatry and Psychopathology

PSYCHONEUROSIS, COMBAT—ANXIETY TYPE. ROBERT B. MCELROY, Am. J. Psychiat. **101**:517 (Jan.) 1945.

McElroy states that psychoneurosis was a problem in the theaters of war and of the various types combat anxiety was considered a major problem among fighting men, being followed by other neuroses, in the order of hypochondriasis, conversion hysteria, mixed types and neurasthenia.

The author indicates that emotional factors—love, morale, discipline, and faith—hastened psychoneurosis, but combat, especially fear of battle, played a major part in the causation of anxiety states. A stable soldier disposed of his fear and relaxed, whereas a psychoneurotic soldier maintained his anxiety after the battle. Factors such as fearful anticipation, inhuman acts and exposure to hazardous conditions were responsible for the psychoneurosis. Minor physical wounds seemed to play no part in the type of neurosis, and a major wound was rarely associated with an anxiety state.

Other factors which were vital in producing the anxiety state were age, rank and preinduction status of the patient. It was noted that the older soldiers were less inclined to have psychoneuroses. The condition was more prevalent among privates than among noncommissioned officers. In more than half the cases of hysteria, in all the cases of neurasthenia and in more than half the cases of mixed type of neurosis symptoms were present prior to induction.

McElroy emphasizes the importance of therapy in cases of combat anxiety, the type of therapy varying with the unit. The basic treatment of an anxiety state was withdrawal of the patient from combat. Severe anxiety states were best treated with sedation, such as intravenous administration of sodium isoamylethylbarbiturate, reassurance, suggestion and segregation. Daily sedation was not advocated because it prolonged hospitalization and did not alter the reality of the existing situation. Other units employed electric shock therapy and cold wet packs. If therapy failed, the soldier was not permitted to return to combat, but was sent to a zone of the interior and reclassified.

BORKOWSKI, Boston.

THE DYNAMISM OF ANTI-SEMITISM IN GENTILE AND JEW. B. BETTELHEIM, J. Abnorm. & Social Psychol. **42**:153 (April) 1947.

Bettelheim bases his discussion of anti-Semitism on observations made while he was imprisoned at the German concentration camps at Dachau and Buchenwald. This study deals with the pathologic interpersonal relations of the Jew and the anti-Semitic person. The author believes that anti-Semitism has to do with the anti-Semitic person himself, with his inner problems and his inability to solve them without recourse to a delusional system, rather than with the Jew and his character. The reactions of the Jew are in defense against the anti-Semitic threat, modified by his inner needs. As time goes on, his defense reactions become the function of inner motivation rather than of outer pressure. He derives some narcissistic gratification from the fact that he is persecuted. The Jew takes advantage of anti-Semitic persecution for bolstering his self esteem in ways which result in his permitting himself the use of devious and ineffective psychologic mechanisms similar to those used by the anti-Semitic persecutors. He projects his own undesirable characteristics onto the anti-Semitic persecutor, thus gaining secondary narcissistic gratification from being persecuted. The Jew attempts to escape anti-Semitism by defending himself through delusional mechanisms. He makes no attempt to test reality, thinking of the Gestapomen in terms of stereotypes. This has the purpose of overcoming anxiety in dealing with the Gestapo and of safeguarding his own self esteem by thinking the other depraved. Because of his inability to test reality, the Jew was unable to work out adequate interpersonal relations with the Gestapoman.

FRANKEL, Philadelphia.

INTERNAL AND EXTERNAL CAUSES OF ANXIETY IN RETURNING VETERANS. ARNOLD EISENDORFER and MURRAY D. LEWIS, J. Nerv. & Ment. Dis. **103**:137 (Feb.) 1946.

Eisendorfer and Lewis discuss the soldiers who carry on successfully while overseas but display symptoms of anxiety, tension or depression after their return to the United States. In cases of this type careful history taking and psychologic exploration reveal the presence of unresolved emotional conflicts stemming from intrafamily tensions experienced in infancy and childhood. During their overseas tour persons of this type are able to withstand successfully the external dangers of war and hardship. Not only does war provide a flight from internal neurotic anxiety, but, being a socially acceptable method of killing and being killed, it furnishes an outlet for sadomasochistic tendencies. With their return to family relationships, the relatively greater internal dangers of their unresolved conflicts are relighted, with resultant neurotic symptoms. Two illustrative cases are presented.

CHODOFF, Washington, D. C.

THE THERAPEUTIC USE OF DREAMS INDUCED BY HYPNOTIC SUGGESTION. MARK G. KANZER, Psychoanalyt. Quart. **14**:313, 1945.

Kanzer reports a case in which dreams induced during hypnosis were used to reveal emotional problems and past events, to provide quick insight into the patient's character and hidden thoughts and to afford a basis for therapeutic discussions. He used his method because the patient was extremely uncommunicative. The dreams showed typical day residues and evidences of both positive and negative transference. Although dreams with elements of hostility to the therapeutist occurred, their presence did not interfere with the cooperation of the patient in the hypnosis.

Kanzer believes that hypnotically induced dreams provide the psychiatrist with unusual opportunities for direct intervention in the unconscious of the patient and offer a field for the experimental study of the dream process. Since the hypnotist is able to provide the topic for the dream, it is possible to secure immediate and relevant data on the meaning of fixed ideas, obsessive practices, fetishes and other symbolic distortions of the neuroses. As compared with the usual course of psychoanalysis, hypnosis has the disadvantage that the pace must be forced to a greater extent. In the active hypnotic technic outlined, the course of therapy is more rigid and provides less for spontaneous adjustment than in psychoanalysis.

PEARSON, Philadelphia.

THE NEGATIVE IN DREAMS. NANDOR FODOR, Psychoanalyt. Quart. **14**:516, 1945.

Freud's statement, "There is no negative in dreams," needs no confirmation. However, negatives do appear in the dream text, and Fodor discusses these.

The negative may decrease tension because the patient could not tolerate the dream thoughts. Here, the negative statement confirms an idea which is simply expressed as not being so. The negative may be an attempt to mitigate the dream thought and to plead for consideration. Sometimes the negative is latent; i. e., the text may state a positive about one thing as a cover for the negative about something else. The negative may not be in the dream but in the associations; it may not be in the dream or in the associations but in a comment or explanation during the narration of the dream, or it may take the form of a doubt in the dream text. In dreams some person other than the dreamer cannot be blamed or accused

of a deed without the dreamer's revealing his own guilt. Neither can the dreamer wonder whether or not something happened in a dream—it did. A true mistake cannot be made in a dream, and a lie in a dream always reveals the truth, as does dreaming of a secret. The dreamer cannot give a false association to a dream without revealing the truth, nor can he pretend in a dream without playing the true role.

Although the dream is the guardian of sleep, it may also have the function of awakening a person.

PEARSON, Philadelphia.

PSYCHOANALYTIC STUDY OF ULCERATIVE COLITIS IN CHILDREN. MELITTA SPERLING, Psychoanalyt. Quart. **15**:302, 1946.

In the 2 cases of ulcerative colitis in children reported here and in similar cases, Sperling found that the mother was extremely ambivalent to the child and had subjected him to early and deep frustrations. The ambivalence was based on strong unconscious destructive impulses toward the child, with a wish to get rid of him. The child reacted with a hostile attitude and an intense need to hold on to the position of a baby, with extremely strong oral and anal sadistic tendencies. He was fixated on an oral sadistic level, owing to early frustration, and was characterized by an extreme inability to tolerate any psychic tension.

The child with ulcerative colitis becomes completely absorbed in the illness because of a narcissistic orientation. The bleeding occurs early and impresses one as being under the child's control. Proctoscopic examination reveals diffuse inflammation and edema of the mucous membrane with transudation of blood. After a number of occurrences, secondary ulceration may take place. Hospitalization increases the condition observed through the proctoscope, the hospitalization representing a traumatic experience by disrupting the narcissistic equilibrium. The child is in a state of permanent frustration, which results in a state of unconscious rage with an irresistible desire for immediate discharge. He attempts through the bleeding to destroy and eliminate the object which has been incorporated sadistically. Although it is the degree of regression that differentiates ulcerative colitis from conversion hysteria and mucous colitis, it is the quantity of sadism that determines the depth of the regression.

Ulcerative colitis is an organ neurosis with pregenital conversion symptoms. The choice of the organ is determined by oral and anal fixations, the colon being the eliminating organ. The anorexia, vomiting, abdominal pain, diarrhea and bleeding represent expressions of and defenses against the aggressive incorporation of the frustrating object.

PEARSON, Philadelphia.

PSYCHOLOGIC INVALIDISM IN THYROIDECTOMIZED PATIENTS. J. RUESCH, CAROLE CHRISTANSEN, L. C. PATTERSON and others, Psychosom. Med. **9**:77 (March-April) 1947.

Ruesch and his associates review the results of a personality study on 43 patients who had been subjected to subtotal thyroidectomy and were selected from the material of the thyroid clinic of the University of California Hospital in such a manner as to constitute a fair representation of the several thousand patients followed in the clinic. The authors believe that patients who have thyroid disease are not entirely normal in their sociopsychologic relations. The female patients may be classified as the normal, or almost normal, type and the hysterical or anxiety type. While surgical procedures are tolerated exceedingly well by the

normal girls, delayed recovery is almost a probability with patients of the hysterical or anxiety type. With men the problem is similar, but instead of hysterical personalities one deals with the dependent type. In preparation for operation, psychiatric interviews might be helpful in detecting deviations in personality of the anxiety, hysterical or dependent type. Therapeutic interviews before and after operation might improve the chances of recovery of patients with personality disorders.

J. A. M. A.

PHANTOM LIMB. J. R. EWALT, G. C. RANDALL and H. MORRIS, Psychosom. Med. **9**:118 (March-April) 1947.

The report presented by Ewalt and his associates is based on observations on amputees during their stay in an Army general hospital. Of a series of 2,284 patients with amputations, 404 had detailed psychiatric study. Phantom sensations were found to occur in practically all persons who were studied carefully. Spontaneous complaints of pain were rather rare in the group. The patients complaining of phantom pain were persons who had psychopathologic manifestations complicating the orthopedic picture. The authors gained the impression that phantom pain is merely the interpretation of a phantom sensation by certain psychopathologic persons. Two cases are reported which support this view. In both the pain tended to come and go with psychopathologic symptoms irrespective of what type of external treatment was carried on.

J. A. M. A.

Diseases of the Brain

DIAGNOSIS AND TREATMENT OF STRICTURES OF THE AQUEDUCT OF SYLVIUS (CAUSING HYDROCEPHALUS). WALTER E. DANDY, Arch. Surg. **51**:1 (July) 1945.

Strictures of the aqueduct of Sylvius are due either to congenital maldevelopment (gliosis) or to healed inflammatory processes within the ventricles. Strictures of the aqueduct invariably produce obstructive hydrocephalus of the lateral and third ventricles. The ventricular dilatation is usually of high degree and more extreme than in cases of tumor of the posterior fossa.

With infants, the diagnosis of stenosis of the aqueduct can be made if (1) the head is greatly enlarged, with a low inion (roentgenograms of the skull may be necessary), and (2) evidence of ventricular obstruction is obtained by failure to recover by lumbar puncture the test dye injected into the lateral ventricle. Ventriculographic studies on infants with stenosis of the aqueduct is not advisable, for the procedure is poorly tolerated.

In older children and adults the enlargement of the head may not be as pronounced as in infants, for in the former the obstruction of the aqueduct is often only partial. A "cracked pot" (Macewen) sign is usually demonstrable, and the inion is either normally placed or lower than normal, in spite of general enlargement of the skull. A ventriculogram may have to be taken for differentiation from cerebellar tumor. In the ventriculogram the outline of the aqueduct, if seen at all, is funnel shaped or triangular.

The best treatment for stricture of the aqueduct is ventriculostomy. Dandy stated that he preferred the subtemporal to the anterior approach, for the following reasons: (1) External hydrocephalus does not follow; (2) it is not necessary to section the optic nerve, and (3) the scar is concealed under the hair. Dandy performed a total of 36 ventriculostomies on 29 patients over 1 year of age. The

operative mortality was low (1 case), and the results were excellent, since, at the time of the report 24 patients were living and appeared to be cured except for defective vision. For the younger group, i. e., the infants, the results were poor. Of 63 patients on whom the operation was performed, 10 died in the hospital, and only 21 were living at the time the survey was made. Most of the survivors had pronounced neurologic or mental defects. In this group the operation is probably worth while only when the head is not over 50 or 52 cm. in circumference at the time of operation.

LIST, Grand Rapids, Mich.

HEMIATROPHY OF THE BODY IN ADULT LIFE. NORMAN REIDER and GLEN S. PLAYER, J. Nerv. & Ment. Dis. **103**:1 (Jan.) 1946.

Reider and Player review the literature on the facial and total hemiatrophies. Hemiatrophies of the latter type have been caused by birth injuries, head injuries in childhood and in adult life, encephalitides of many types, vascular lesions and tumors. The etiologic factor is obscure in some cases of the syndrome.

The first case reported by the authors was that of a soldier aged 24 who was hospitalized, after considerable combat experience, for trench feet. At the time of his discharge to duty, it was noted that the arm and leg on the right side were smaller than those on the left. Except for the atrophy, the neurologic status was entirely normal. A pneumoencephalogram made six months after onset of the atrophy revealed bilateral internal hydrocephalus, the left ventricle being larger than the right.

The second case was that of a soldier aged 22 who was hospitalized because of pain and stiffness of the right leg and a history during the previous year of not having been able to run as fast as was usual for him. Examination revealed slight atrophy of the right side and a positive Babinski sign on that side. Pneumoencephalographic examination revealed dilatation of the left lateral ventricle, especially the middle portion.

The authors believe that the cerebral origin in their cases is established by the pneumoencephalographic findings. In cases of facial hemiatrophy and of atrophy of an extremity of unknown cause, air studies should be made, since the condition may also be of cerebral origin.

CHODOFF, Washington, D. C.

DIAGNOSTIC IMPORTANCE AND MECHANISM OF ABNORMAL POSITIONS OF THE HEAD IN CASES OF SURGICAL LESIONS OF THE POSTERIOR FOSSA. T. FRACASSI, R. J. BABBINI and R. GRAZIANO, Rev. argent. neurol. y psiquiat. **10**:49 (March) 1945.

Fracassi, Babbini and Graziano report 4 cases illustrating the clinical significance of alterations in posture of the head associated with lesions of the posterior fossa. The first case was that of an unverified lesion of the posterior fossa, probably arachnoiditis. The patient had attacks of severe headache with opisthotonos. In the second case the presence of arachnoiditis in the posterior fossa was verified. The only focal sign before operation indicating the existence of the lesion in this region was extension of the head during bouts of cephalalgia. In the third case a glioma in the pontobulbar region was observed at autopsy; there were diffuse signs during life suggesting multiple sclerosis, for which the patient was treated for a time. The only sign pointing to the probable presence of a tumor in the posterior fossa was persistent anterior flexion of the head without headache; this

position of the head was considered the result of an attempt to avoid compression of the enlarged medulla against the posterior border of the foramen magnum. The fourth case was that of a verified cystic tumor in a cerebellar hemisphere. Retraction of the head was observed during exacerbations of headache. This fact pointed to a tumor of the posterior fossa, even though the patient had other complaints which suggested localization in the parietal lobe. The authors believe that the anterior flexion and lateral deviation of the head in cases of tumors of the posterior fossa are attempts to compensate for alterations in the position of the medulla. Extension of the head is occasionally an attempt to minimize pain and at other times is a reflex decerebrate rigidity resulting from compression of the brain stem.

SAVITSKY, New York.

ENCEPHALOPATHIES ASSOCIATED WITH ACROMEGALY. LEON LEVIT, Rev. Asoc. méd. argent. **59**:615 (June 15) 1945.

Levit reports 3 cases of cerebral diplegia in patients 14, 15 and 17 years of age, respectively, who showed acromegalic features (large face and extremities and separation of teeth). In only 1 case was roentgenographic examination mentioned as showing no abnormalities. All the patients were mentally retarded. No clear physiologic relation between the acromegalic changes and the cerebral diplegia was noted.

SAVITSKY, New York.

HERPES ZOSTER ENCEPHALITIS. W. SCHMIDT, Deutsche med. Wchnschr. **71**:308 (Dec. 13) 1946.

Schmidt relates that a man aged 56, who had herpes zoster in the region supplied by the first branch of the trigeminus nerve, died as a result of a simultaneously existing meningoencephalitis, which, however, had not been recognized. Drowsiness was one of the most noteworthy signs. In this respect the condition resembled von Economo's lethargic encephalitis. The herpes zoster, like all other forms of virus encephalitis, is chiefly a polioencephalitis. The medulla and pons were most severely involved in the case under consideration. Further observations will be necessary to ascertain whether this localization of the inflammatory foci is typical of zoster encephalitis. The type of the microscopic changes is largely identical with those observed in other forms of virus encephalitis, particularly epidemic encephalitis, except that the substantia nigra is less severely involved than in epidemic encephalitis.

J. A. M. A.

CLINICAL PICTURE OF RETROBULBAR NEURITIS ASSOCIATED WITH INTRACRANIAL TUMORS. S. LARSSEN and B. NORD, Nord. med. (Hygiea) **34**:1059 (May 2) 1947.

Larssen and Nord warn that in cases of tumor in the anterior or middle fossa reduced vision and central scotoma, with or without papilledema or atrophy of the optic nerve, constitute an early symptom the diagnostic value of which is frequently not recognized. The symptom is often attributed to sclerosis disseminata, and surgical intervention is thus delayed. Especially in cases with a protracted course, encephalographic study is indicated. Eight personal cases, namely, 2 of meningioma of the olfactory groove, 2 of suprasellar meningioma, 1 of meningioma originating in the lesser wing of the sphenoid bone, 1 of adenoma of the hypophysis, 1 of tumor of the hypophysial duct and 1 of subfrontal meningioma, are described in detail. In all cases diagnostic difficulties were presented and retrobulbar neuritis

was prominent. Spontaneous improvement in vision suggested the erroneous diagnosis of sclerosis disseminata in the first 4 cases; the neuritis was ascribed to anemia in 1 case, to possible cerebral trauma in 1 case and to arteriosclerosis in another; in only 1 case was cerebral tumor diagnosed without preceding misconception.

J. A. M. A.

Diseases of the Spinal Cord

End Result Study of Intervertebral Disk. R. E. Lenhard, J. Bone & Joint Surg. **29**:425 (April) 1947.

Lenhard studied 843 patients operated on for removal of an injured intervertebral disk. Five hundred and sixty-six (67 per cent) of the patients were men and 277 (33 per cent) were women. The operation afforded good results in 67.5 per cent of the patients examined. In those not examined but surveyed by questionnaire, apparently good results had been obtained in 59.5 per cent. In a series of 147 who returned for examination, 20 patients required multiple operations for recurrences. Roentgenographic interpretations are not reliable for accurate localization of the lesion.

J. A. M. A.

Anatomophysiologic Aspects of Injuries to the Intervertebral Disk. V. T. Inman and J. B. de C. M. Saunders, J. Bone & Joint Surg. **29**:461 (April) 1947.

Inman and Saunders divide their patients with injuries to the intervertebral disk into three categories. 1. Patients with backache and local signs and symptoms of injury to the vertebral ligamentous structures with radiating pain, deep in character, extending down one or both extremities. Such patients should be considered as having mechanical derangement of the spine and should be treated by immobilization and support. 2. Patients whose symptoms are identical with those of the first group but who, in addition, give definite evidence of compression of the nerve roots. Pain is relatively as prominent a phenomenon as the evidence of compression. Treatment should be directed toward the relief of pressure in order to prevent permanent damage to the affected nerve root. After removal of the herniated or displaced disk, additional measures are necessary to protect the injured and still painful, ligamentous structures. 3. Patients whose symptoms of backache may be mild or lacking and in whom radiation of pain to the extremity is not extensive but signs of compression of the nerve roots are prominent. There are muscular weakness with atrophy, loss of sensation over the appropriate dermatome and reflex changes. Relatively few cases of herniated intervertebral disk fall in this category, but many cases of tumor of the spinal cord do. Removal of the pressure agent and decompression of the nerve may be followed by almost complete dramatic recovery.

J. A. M. A.

Some Unusual Findings in Epidemic of Anterior Poliomyelitis. J. H. Nicholson, New England J. Med. **235**:214 (Aug. 15) 1946.

During March and April 1945 a southern Army post experienced a short outbreak of anterior poliomyelitis. Within a period of twenty-five days 18 patients with poliomyelitis were admitted to the hospital. All the patients were soldiers;

all but 3 were between 18 and 21 years of age. The cases had some unusual features. Bulbar involvement occurred in 3 patients. There were 3 deaths, all of patients with bulbar involvement and all caused by failure of the respiratory center. A large percentage of the patients failed to show the cellular response of the spinal fluid usually associated with poliomyelitis. The albuminocytologic disassociation described in the Guillain-Barré syndrome was found in 3 of the 18 patients of this series.

J. A. M. A.

STAPHYLOCOCCAL SPINAL OSTEOMYELITIS WITH ACUTE EXTRADURAL ABSCESS. GUY BLACKBURN and R. P. JEPSON, Brit. M. J. **2**:297 (Aug. 31) 1946.

Extradural abscess, if not treated surgically, is invariably fatal, and the mortality rate in cases in which operation is performed has been estimated as 35 per cent. Surgical treatment consists in incision by an extensive laminectomy and drainage either by repeated packing or by insertion of a drainage tube of large bore; the consequence of this procedure has been prolonged hospitalization while the spinal cavity collapsed or a sinus closed. Early diagnosis would permit primary closure in most cases. Jepson reports the case of a man aged 36 in which incision and drainage were undertaken immediately; the wound was completely closed with layered surgical gut sutures, and a small tube of penicillin was brought to the lower end of the extradural abscess by means of a separate stab incision. The patient started vigorous physical therapy on the tenth postoperative day, was walking at the end of the first fortnight and was free from symptoms and ready to return to duty at the end of two months.

ECHOLS, New Orleans.

ENDOCRINE AND MENTAL DISORDERS IN ACUTE ANTERIOR POLIOMYELITIS. R. LUFT and R. MÜLLER, Acta med. Scandinav. **127**:448 (May 10) 1947.

Luft and Müller examined 66 persons, of whom 54 had had acute poliomyelitis during the period of 1944 to 1945 and 12 during the period of 1934 to 1936; endocrine disorders were observed in 20 of these patients, all of whom were women. The endocrine disorders varied to a striking degree: There were menstrual disorders in 8 patients, changes of body weight in 11, disturbances of sexual and bodily development in 3 and increased thirst in 3. Decreased dextrose tolerance was demonstrated in 14 patients on whom tests were performed. Hypertrichosis was present in 24 patients. It was commonest in patients with pronounced paresis of the extremities. In several of these patients the hypertrichosis was probably a manifestation of a trophic disorder following injury of the peripheral neuron. Mental disorders were observed in 36 of the 66 patients. Those who had poliomyelitis as adults showed mental disorders of the organic neurasthenic type. In 4 of the 7 patients who had the disease during childhood a change of personality was noticed. The frequency of mental disorders was the same for persons who had the disease in childhood and for those who had it in adult life. Physical invalidity was of little importance in the development of mental disorders. There was no correlation between clinical signs of encephalitis during the acute stage of poliomyelitis and endocrine or mental disorders in the after-stage. Endocrine and mental disorders appeared independently of each other. The pathoanatomic basis of these endocrine and mental disorders is obscure; they might be considered manifestations of inflammatory changes in the hypothalamus.

J. A. M. A.

Treatment, Neurosurgery

ELECTRONARCOSIS: ITS APPLICATION AND THERAPEUTIC EFFECT IN SCHIZOPHRENIA. ESTHER BOGEN TIETZ, GEORGE N. THOMPSON, A. VAN HARREVELD and C. A. G. WIERSMA, J. Nerv. & Ment. Dis. **103**:144 (Feb.) 1946.

Electronarcosis is a method of applying alternating current to the brain for prolonged periods, producing a controlled state of unconsciousness, preceded by a convulsion. The authors used this procedure in treating 47 schizophrenic patients with predominantly paranoid symptoms. The instrument used delivered a 60 cycle alternating current of a predetermined amperage, independent, within certain limits, of the resistance of the head and electrodes. A current of 160 milliamperes is usually selected for the first treatment. When the current is switched on, there is an immediate tonic flexion of the extremities, followed after ten to fifteen seconds by extension of the legs. An initial short cardiac arrest is followed by a period during which the heart beats slowly, and often irregularly. After thirty seconds the high initial current is rapidly decreased to from 60 to 75 milliamperes, and there are weak clonic twitches for about ten seconds. Respiration starts after application of current for forty-five to sixty seconds. The current is slowly increased at the rate of 5 milliamperes per fifteen seconds until the appearance of inspiratory stridor. The electronarcosis is continued for seven minutes. Treatment is given three times a week for at least four weeks. If there has been no improvement after twenty treatments, no more are given.

One month after termination of treatment 19 patients had a complete recovery; 16, social recovery, and 7 failed to show improvement. Of 13 patients with gradual onset of the disease, 6 recovered and 5 had social recoveries. Four of the 7 patients who failed to benefit from treatment were included in the group with illnesses of more than two years' duration. Six months after treatment 6 patients in the group with satisfactory results had relapses. In 1,400 treatments, no serious complications were encountered.

CHODOFF, Washington, D. C.

HISTAMINE THERAPY IN MULTIPLE SCLEROSIS. H. R. CARTER, J. Nerv. & Ment. Dis. **103**:166 (Feb.) 1946.

Carter treated 20 patients suffering from multiple sclerosis with histamine according to the technic of Horton. An infusion prepared by adding histamine diphosphate, 2.75 mg., to 250 cc. of isotonic solution of sodium chloride was administered intravenously at the rate of from 30 to 90 drops per minute. The patients received from ten to forty such courses. Seventeen patients failed to show any improvement, while 3 showed temporary improvement of less than six months and then relapsed.

In disagreement with the theory of the allergic cause of multiple sclerosis, Carter points out the rarity of this condition in childhood as compared with the frequency of such known allergic disorders as bronchial asthma, atopic rhinitis, hay fever and dermatitis. Moreover, other evidences of allergy are rare in multiple sclerosis, although food allergy is occasionally found in the background. Again, although there are acute remissions and exacerbations in the course of multiple sclerosis, these do not occur with the dramatic suddenness of allergic diseases.

CHODOFF, Washington, D. C.

HYDROCEPHALUS: A CONTRIBUTION RELATED TO TREATMENT. OLAN R. HYNDMAN, J. Neurosurg. **3**:426 (Sept.) 1946.

Hyndman previously reported on 4 cases of hydrocephalus treated by choroid plexotomy. The operation consists in evulsion of the choroid plexus at the glomus, thus establishing communication between the lateral ventricles and the subarachnoid space through disruption in the laminas of the choroid plexus. In the author's opinion, an opening in this region is less likely to be obliterated by normal healing processes.

He again reports on the 4 cases and adds a fifth. In 3 cases of congenital hydrocephalus treatment was with ventriculocisternostomy. In all cases the hydrocephalus was relieved. In 2 cases death occurred from nonsurgical causes and in 1 case the patient was still in excellent condition at the time of writing, with no increase in size of the head. In 1 case, that of a man with chronic arachnoiditis and resulting hydrocephalus, the patient was treated in the same way and two months later had normal ventricular pressure. In the fifth case, that of a 7 year old girl with hydrocephalus following pneumococcic meningitis and adhesive basal arachnitis, choroid plexotomy was performed. Twenty-one months later (January 1946) she had normal spinal fluid pressure with complete regression of her previous papilledema.

Hyndman concludes that this procedure provides a simple and most effective means of treating congenital and acquired hydrocephalus, both of the communicating and of the noncommunicating type.

TOZER, Philadelphia.

A SAFE METHOD OF DETOXICATING THE ACUTELY ILL ALCOHOLIC. Z. M. NASON, Quart. J. Stud. Alcohol **8**:43 (June) 1947.

Nason describes a tapering-off method of detoxicating the acutely ill alcoholic patient which aims at combating mental depression, a low chloride level of the blood, low blood sugar and dehydration. The patient is given, in a single intravenous injection, 50 cc. of a 50 per cent solution of dextrose, 10 U. S. P. units of regular insulin and 1 cc. of a preparation of the vitamin B complex. This injection is repeated at four hour intervals for three to five doses. After the final injection, the process of detoxication is begun. The patient is given 2 ounces (60 cc.) of whisky diluted with 6 ounces (175 cc.) of water. This medication is repeated at three hour intervals for a total of five doses. After the fifth dose, a six hour interval is allowed. The same amount of whisky in the same dilution is then given and the amount repeated at six hour intervals for a total of three doses. In addition, the patient is given 1 teaspoonful of table salt, dissolved in a glass of water, one hour after the second, and again one hour after the fourth, dose of whisky. During the intervals between the drinks of whisky the patient is urged to drink black coffee containing 1 or more teaspoons of sugar. This part of the treatment is modified if the patient is found to be diabetic. The procedure can be modified to be carried out by the layman, when medical supervision is not available, by the omission of the injections of dextrose, insulin and vitamin B complex.

FRANKEL, Philadelphia.

ELECTROCONVULSION THERAPY IN THREE HUNDRED AND ONE PATIENTS IN A GENERAL HOSPITAL. DALTON E. SANDS, Brit. M. J. **2**:389 (Aug. 31) 1946.

Sands analyzed a series of 301 cases of mental disorders treated with induction of electric convulsions, particularly with regard to selection of cases and response to therapy. There were 80 cases of neuroses, 134 cases of minor psychotic depres-

sion and 87 cases in which symptoms of both conditions were manifested. Recovery after twelve months was reported in 5 of 13 cases of obsessional neurosis, in 4 of 8 cases of depression with obsession, in 2 of 18 cases of anxiety neurosis, in 10 of 17 cases of depression with anxiety, in 3 of 20 cases of hysterical neurosis, in 5 of 24 cases of depression with hysteria and in 44 (60 per cent) of 73 cases of depression. Of the 221 cases of depression, relapse occurred from six months to four years after treatment in 64 (28.9 per cent).

With regard to the mode of action of convulsion therapy, Sands states that it is difficult to escape the conclusion that a variable degree of disintegration and regression of personality is required, sufficient to disorganize the recently acquired habits of feeling and thinking; then, by employing long-established reflexes and patterns of behavior, the personality readjusts at a slightly lower level, but does not necessarily stay there.

The number of convulsions required must be adapted to the type of illness and the severity in each case. Whether or not psychotherapy is used in conjunction with convulsion therapy depends on the type of depression and how far it has progressed.

With respect to the occurrence of fractures during treatment Sands writes, "The symptomless character of most compression fractures occurring after convulsion therapy, the absence of subsequent disability over ten years and the finding of similar fractures in 10 per cent of epileptics have allayed much of the anxiety felt in the matter." Convulsion therapy had no ill effects on 2 pregnant patients. No late fits suggestive of induction of an epileptic habit were known to have occurred in the author's series.

ECHOLS, New Orleans.

GLOSSOPHARYNGEAL NEURALGIA TREATED BY TRANSTONSILLAR SECTION OF THE NERVE. C. P. WILSON and D. MCALPINE, Proc. Roy. Soc. Med. **40**:82 (Dec.) 1946.

The glossopharyngeal nerve of a woman aged 47 was exposed in the tonsillar fossa after removal of the right tonsil; it was divided and 3/8 inch (0.95 cm.) removed, with relief of typical glossopharyngeal neuralgia.

The syndrome is characterized by paroxysmal pain referred to the back of the tongue and the tonsillar area, radiating to the ear and having a trigger zone. The transtonsillar approach is recommended if the trigger zone exists in the tonsillar area, the intracranial approach being a last resort if the newer method fails to give relief. Another indication for the use of this approach, which is described by the authors as surgically simple, is failure to demonstrate a primary condition causing the neuralgia such as an elongated styloid process pressing into the tonsillar bed.

BERRY, Philadelphia.

Society Transactions

PHILADELPHIA NEUROLOGICAL SOCIETY

Robert A. Groff, M.D., *Presiding*

Regular Meeting, Oct. 25, 1946

Case of Extrapyramidal Disease of Twelve Years' Duration: A Follow-up Study. DR. BERNARD A. HIRSCHFIELD and DR. JOSEPH C. YASKIN.

J. F., a man now aged 54, had onset of attacks in 1935 characterized by an irresistible twisting of the head to the right. After a few months the head became maintained more or less continuously in a position with the chin turned to the right and slightly down. In this position the left sternocleidomastoid muscle and the right deep cervical muscles were felt to be in spasm. The diagnosis of tonic torticollis was made. All peripheral causes of the condition were ruled out. The question then became: Was this disorder of extrapyramidal origin, or was it psychogenic?

Psychiatric study revealed a seemingly well adjusted person with no obviously difficult presenting situation. There was apparently little indication for psychotherapy. A series of artificially induced fever treatments was given, after which there was complete relief from the torticollic spasms.

The patient, when seen again in July 1946, was found to have well defined signs of parkinsonism, including typical facies, increased muscular tonus, pill-rolling tremor of the right thumb, poverty of small movements and absence of swinging of the right arm in walking.

There is a suggested possible relation between the spasmodic torticollis and the later developing parkinsonism in this case. This observation may corroborate the opinions expressed by Foerster, Brain and others that spasmodic torticollis is in some cases the result of organic involvement of the extrapyramidal system.

DISCUSSION

DR. JOSEPH C. YASKIN: The reason we decided to present this case is that the patient had torticollis twelve years ago, recovered after artificial typhoid treatment and then was fairly well for about ten years, when there developed what now appears to be a parkinsonian syndrome.

Twelve years ago, however, the torticollis was not a spastic torticollis, it was a tonic torticollis. Acquired tonic torticollis always presents a serious diagnostic problem. In most cases it is not organic in type. Whatever may be thought about spastic torticollis, tonic torticollis is usually functional, or due to local disease of the eleventh nerve or of the sternocleidomastoid and associated muscles, possibly Pott's disease which is overlooked for a number of months.

Whether in this case tonic torticollis was associated with acute encephalitis that was overlooked, and the patient's present condition is a sequel to that encephalitis, or whether at the time that he had the tonic torticollis he was psychoneurotic, I do not know.

All forms of torticollis cannot be viewed as psychogenic. Most, in my opinion, are of organic origin. In many cases, in the course of time, additional physical abnormalities develop. Moreover, from a practical standpoint, spastic torticollis is organic in that it is not amenable to any form of treatment, including psychotherapy and neurosurgical procedures.

As with many of the tics, a large number of the compulsive reactions are poorly understood conditions which are not wholly psychogenic at any time. They probably are fixed physiologic disturbances, and they all have one thing in common: they resist treatment, and the prognosis is poor. That goes for all forms of therapy, including prolonged psychoanalysis.

DR. FRANCIS C. GRANT: From about 1923 to 1928 Dr. Frazier was much interested in the problem of spastic torticollis and operated on a number of patients —I should think from between 25 to 30. My colleagues and I see a certain number of those patients who now have dystonia. I was interested in noting that neither Dr. Yaskin nor Dr. Hirschfield mentioned dystonia or suggested that the spastic torticollis was possibly an early dystonia; at least half a dozen patients on whom Dr. Frazier operated now have full-fledged dystonia.

My observation is that if a number of patients who have spastic torticollis are carefully examined one is likely to find evidence of dystonia in other muscular structures. That, to my mind, is one of the pitfalls into which the surgeon can easily tumble if he operates on such patients without careful examination. I feel exactly as Dr. Yaskin does, that is, that spastic torticollis does not lend itself readily to surgical treatment. My impression, which I cannot back with any figures, is that probably in 75 per cent of the cases in which Dr. Frazier operated the outcome has been unsuccessful.

DR. CHARLES RUPP: The patients mentioned by Dr. Grant remind me of one seen several years ago at the Philadelphia General Hospital. A young man in his late twenties had onset of a spasmodic torticollis shortly after his wedding. Several years prior to this he had been involved in a homosexual liaison and then became engaged. A few days before the wedding his fiancée eloped with her sister's boy friend, and the patient married the jilted sister. The marriage was not a happy one. Because of the emotional factors, it was thought that his torticollis was psychogenic, and he had an orthodox psychoanalysis, without benefit. For a long time we were uncertain whether his condition was psychogenic or of organic origin, but within a year he manifested full-blown dystonia musculorum deformans, which was extremely disabling. His movements were so violent that he fell out of bed at night. Dr. Groff extirpated area 6 a of the cortex, without any appreciable effect. The patient became depressed because of this disability, and some one who was then treating him recommended shock therapy. He died within a few minutes after receiving his first electric shock. There was no autopsy.

DR. JOSEPH C. YASKIN: In reply to Dr. Grant, I might say that in this presentation mention was made of spastic torticollis being a fragment symptom of dystonia.

DR. HENRY T. WYCIS: I was interested in Dr. Grant's comment on the Frazier cases. I believe the operations were posterior rhizotomies, were they not?

DR. FRANCIS C. GRANT: Yes.

DR. HENRY T. WYCIS: In 2 cases I tried Dandy's method of sectioning the anterior roots of the first, second and third cervical nerves and both spinal accessory nerves intradurally. In 1 case it has now been two years since operation, and the patient is entirely free of any spasmodic movements. In the second case there was probably about 50 per cent improvement.

I should like to hear about Dr. Groff's and Dr. Grant's experiences in their recent surgical treatment of these patients.

DR. ROBERT A. GROFF: Do you want to answer that, Dr. Grant?

DR. FRANCIS C. GRANT: I do not think we have operated in a case of torticollis in ten years. We were so discouraged by seeing the results of operation in the

cases of dystonia ten, fifteen or twenty years ago that we abandoned it as a procedure. My impression is that there have been no cases of spastic torticollis in which operation was done. Do you remember any, Dr. Groff?

DR. ROBERT A. GROFF: I do not remember any.

DR. HENRY A. SHENKIN: We cut the sternocleidomastoid muscle in 2 cases, as I recall.

DR. FRANCIS C. GRANT: I prefer psychotherapy.

DR. JOSEPH C. YASKIN: Dr. Groff, I should like to mention a patient who was first observed several years ago. He was a man with spastic torticollis who required a large collar to be comfortable. I put him through a period of narcosis, such as it was in those days, and then a partial analysis; and at the end of three months he was completely free from the spastic movements. He went back to Pittsburgh and then wrote to me, "For the last few weeks I have had no movement in my neck, but I would much rather have it because I now have terrific anxiety; I can't sleep, and when I sleep I have nightmares. I would much rather have my torticollis and have the movements of the neck."

Later his anxiety disappeared, and his torticollis recurred. Nevertheless, I do not believe the whole disturbance was psychogenic in that case. I think there is something missing in understanding of such cases.

Remissions occur in many organic diseases. I think Foerster understood this condition best. He did not claim that the torticollis is entirely organic, nor did he believe that it is entirely psychogenic; it is a mixture of the two.

Postoperative Period of Survival of Twenty-Five Patients with Oligodendrogliomas of the Brain. DR. HENRY A. SHENKIN, DR. FRANCIS C. GRANT and DR. JOHN H. DREW (by invitation).

Six of the series of 25 oligodendrogliomas (24 per cent) were located principally within a lateral and/or the third ventricle. Seventeen of the 19 oligodendrogliomas growing in the parenchyma of the brain were located in one or the other, or in both, frontal lobes.

The ventricular group of tumors had a short preoperative course (average, seven and one-half months), and the symptoms were those of increased intracranial pressure. The hemispheric group of tumors had a longer preoperative duration of symptoms (average, thirty-five months). The history generally was one of focal symptoms, often jacksonian seizures, preceding the symptoms and signs of intracranial pressure.

The average period of survival after operation in this series of patients was less than two years. Of the 5 patients with ventricular tumor who survived operation, only 1 is still alive and well (at the end of fifty months). The average survival period of the remaining 4 patients was twelve and one-half months, with a range of two to twenty-three months. Fifteen patients with hemispheric tumor recovered from operation, but only 4 are still alive, ten to thirty months later. The remaining members of this group survived an average of twenty-two months, with a range of five to forty-eight months.

DISCUSSION

DR. FRANCIS C. GRANT: I am always interested in the grouping of the various types of gliomas and the efforts that are made to learn the life histories of these tumors. It is particularly significant in this report that the tumors fall into two definite groups—those within the ventricles and those within the cerebral hemispheres.

However, in these days my only feeling about cerebral tumors—aside from spongioblastoma and the medulloblastoma, with which the neurosurgeon must admit he is defeated from the start—is that the type of glioma does make as much difference as the type of surgical intervention directed against the tumor. Neurosurgeons in these days have certainly become much bolder, if you want to put it that way, in their attacks on tumors. They have much less respect for the brain as a functioning unit than had we older surgeons.

I was quite upset at the last meeting of the Cushing Society. A man there from a good clinic in the Middle West had a patient with an astrocytoma, which is a benign tumor, in the right temporal lobe. He exposed the tumor, made a small incision through the cortex, put in a suction apparatus and simply sucked out as much of the tumor as he could. I do not believe that that is good neurosurgical practice. It is my impression that in the case of a tumor of that kind in the right temporal lobe, which is a silent area of the brain, the proper procedure would be a lobectomy and removal of the temporal lobe with the tumor. With that kind of surgery, the rapidity of growth of the tumor will not make much difference, provided the surgeon can get around the tumor. What will determine the effect of neurosurgical procedures in the future will be the radical attack that the surgeon makes on the tumor.

If we had the operations on this series of oligodendrogliomas to do again, in the light of experience with radical procedures which we are accumulating, we should cut about these tumors, whether on the left side of the brain or on the right, and we should probably have a better postoperative morbidity rate than we had. Our mortality might be higher, but I think we should be able to preserve a greater number of patients over a longer time in a reasonably satisfactory condition, so that they could live a happy life.

I am quite convinced that in the future much less emphasis will be laid on the type of tumor than on the surgical procedure which is done to extirpate that tumor. The wider the excision, the more radical the operation; the better the results will be, regardless of the type of glioma involved.

Dr. Robert A. Groff: This is an interesting paper, and I agree with Dr. Grant in his statement about the surgical methods to be used with these tumors. We neurosurgeons are becoming more radical as our technic has improved, and in all probability this will continue.

Dr. Shenkin's statements concerning the dissemination of these tumors are extremely interesting. In a study which I made on the extension of gliomas, the one type which I observed to extend for great distances was the oligodendroglioma. This was particularly noticeable when the tumor was adjacent to the ventricle and extended by long projections over the surface of the ventricle. The oligodendroglioma was disseminated in this manner more than any other glioma. This tendency was brought to my attention by a pinealoma which had infiltrated the walls of the entire ventricular system. Thus, such tumors cause death because they rapidly obstruct the aqueduct of Sylvius after the dissemination begins.

Dr. Michael Scott: The group of cases which the authors have presented illustrates the problem of prognosis with respect to convulsions. Like the meningiomas, a complete operation can be done and the tumor entirely removed, and yet in a large percentage of cases—73 per cent in their series—convulsions may occur after operation. This demonstrates how guarded the prognosis must be concerning the occurrence of convulsions even after a successful operation.

I should like to ask Dr. Shenkin the number of cases he observed in which the type of calcification said to be characteristic of oligodendroglioma was present.

DR. FRANCIS M. FORSTER: I should like to ask a question which is not quite germane to the problem. I notice that the authors had vascular patterns outlined in one of their tumors. I should like to ask whether they have been using arteriography extensively in determining the type of tumor prior to operation. Again, have they had any success with that method?

DR. GABRIEL SCHWARZ: I was extremely interested in Dr. Shenkin's statement that the oligodendroglioma seemed to show more rapid growth after operation. I wonder whether in this series he had an opportunity to compare histologically the tumor when removed at operation and the tumor tissue obtained several years later, at the death of the patient. Was there any histologic difference in the tumor after operation which would explain the increased rate of growth following surgical intervention?

DR. HENRY A. SHENKIN: Dr. Scott inquired concerning the number of tumors in which calcification was noted. In 13 of the 25 tumors, a little over 50 per cent, calcification was apparent in the roentgenograms.

Dr. Forster asked about arteriography in the differentiation of gliomas. Dr. Drew has made a considerable number of attempts to outline vascular patterns in these gliomas. Perhaps he will say something about that. I do not think we have had sufficiently wide experience on which to draw any general conclusions.

Dr. Schwarz asked about the examination of the tumor at reoperation. I had the opportunity to examine 5 of the patients one or two years after the original operation and was unable to observe any changes in the tumor which would indicate more rapid growth after the original surgical intervention.

DR. JOHN H. DREW: We have made arteriographic studies on patients with mass lesions, but so far we have not been able to determine vascular characteristics of any type of tumor. Oligodendrogliomas and meningiomas tend to be vascular. We have made several arteriograms of patients with meningiomas in which the roentgenograms were no more characteristic than those of patients with glioblastoma, which shows no vascular changes at all. The chief value, in addition to localization, is to show that some tumors are more vascular than others.

An Electromyographic Study of Defects of Neuromuscular Transmission in Poliomyelitis. DR. ROBERT HODES (by invitation).

Fourteen patients were examined electromyographically 4.5 to 11.5 years after the onset of symptoms of acute poliomyelitis. The motor nerves supplying the muscles of the leg, foot, forearm and hand were stimulated percutaneously with maximal shocks, and the muscle action potentials were recorded by means of surface electrodes.

Single maximal shocks produced potentials which, except for being small, were otherwise normal. The muscles of the leg were more seriously paralyzed than were those of the foot. The muscles innervated by the peroneal nerve suffered a greater loss of power than did those supplied by the tibial nerve.

Brief repetitive nerve stimulation evoked an abnormal electromyogram. The second and succeeding similar maximal stimuli activated fewer muscle fibers than did the first shock in the series. The defect causing this type of response was shown to be at the neuromuscular junction. Neostigmine (1.0 to 1.5 mg. intramuscularly) reduced the block at the junction.

These findings thus reveal a peripheral derangement of the neuromuscular system in poliomyelitis. They raise, but do not attempt to answer, the important question of how these defects originate—whether by extension of the morbid

process from the central nervous system or other parts of the body to the junctional region, or as a direct result of the action of the virus on the synapse of nerve with muscle.

DISCUSSION

DR. GEORGE D. GAMMON: Dr. Hodes has presented an important series of observations which throw some light on the mechanisms of the weakness in poliomyelitis. I should like to ask several questions about the interpretation of the results.

What is the complete evidence that the results were due to myoneural block? What is the explanation of the shortening of the duration of the action potential? Is there any evidence that nerve fibers may drop out rather than a block occur at the myoneural junction? What is the percentage of recovery with neostigmine in cases of this condition as compared with that which occurs in cases of myasthenia gravis?

It is my impression from Dr. Hodes' records that there was much less recovery in this condition than in myasthenia. Clinically, neostigmine has been tested in cases of poliomyelitis, without obtaining as much return of voluntary power as may be seen in cases of myasthenia. The same is true of other varieties of partial peripheral denervation. This fact has been made the basis of a diagnostic test for myasthenia gravis.

Dr. Hodes has presented a beautifully worked out series of experiments.

DR. GABRIEL SCHWARZ: It is known that quinine increases the muscular weakness of myasthenia gravis and that the same drug produces a reduction in symptoms of myotonia. I wonder whether Dr. Hodes tested the effect of quinine on the electrical reactions of the muscle in the cases of poliomyelitis.

My other question is whether he measured the return of the electrical activity to the muscle after tetanic stimulus. When does this activity return, and what is the length of time necessary for the response to the after-stimulus to regain the same level as that to the initial stimulus?

DR. ROBERT HODES: In answer to Dr. Gammon's questions: My purpose in using neostigmine was not primarily to determine its efficiency, or lack of it, as a therapeutic agent but, rather, to elucidate some of the mechanisms responsible for the fatigue which is so evident during electromyographic observations. I considered increasing the dose of neostigmine, as by using repeated small doses or giving the drug by mouth over a long period; but it seemed that such an extensive study of drug action should be carried out in conjunction with some one with adequate clinical experience. I believe I have demonstrated the existence of a peripheral defect in poliomyelitis, and now it is important to examine this phenomenon more closely, especially clinically.

There is no doubt that one is concerned here with the participation of a smaller number of active muscle fibers excited by the second and succeeding shocks than by the first. Harvey and Masland have discussed thoroughly the evidence leading to such an interpretation. At least part of the loss of muscle fibers, I believe, must be due to a neuromuscular block, since neostigmine restores the potentials to normal. The possibility also exists, as Dr. Gammon suggests, of a failure of nerve fiber action, but I have no evidence for it as yet.

DR. GEORGE D. GAMMON: Do you regard the effect of neostigmine as conclusive evidence of a myoneural block?

DR. ROBERT HODES: The evidence in this study suggests a defect at the myoneural junction, since there is no indication that neostigmine affects the peripheral

nerve directly. Furthermore, in the single motor unit I showed that there was a progressive decline of the potential, rather than an abrupt fall to zero. In this case, at least, the failure cannot be due to a pathologic process in the nerve.

I did not study the clinical return of power in poliomyelitis. There were no facilities at my disposal for that. Neither did I study the effect of quinine.

The size of the potential returns to normal after fatigue. The curve for recovery is simple: The greater the time elapsed since stimulation, the greater is the potential. In one experiment, I recall, the potential reached its original value about 2.5 seconds after the end of a one second period of tetanus. Recovery also took place in the motor unit if one allowed sufficient time to elapse after the first contraction. At intervals between the end of muscular activity and full recovery, the size of the potential was larger than the last spike of the potential which occurred with tetanus but smaller than that which occurred with complete recovery.

Electromyographic Studies on Cats After Section and Suture of the Sciatic Nerve. JAMES G. GOLSETH and DR. JAMES A. FIZZELL.

In this investigation, electromyographic data were obtained from the tibialis anticus muscles of 6 cats after section and immediate suture of the left sciatic nerve high in the thigh.

The electromyographic apparatus consisted of a three stage, push-pull preamplifier; a cathode ray oscilloscope; a power amplifier, and a loud speaker. The three electrodes required to make contact with the cat were (1) an electrocardiograph plate ground electrode, (2) a copper strip indifferent electrode and (3) a "vinylite"-insulated needle electrode, which was inserted into four chosen areas of the given cat's muscle according to a sampling scheme.

One-half hour before each examination the designated animal was placed in a cloth sleeve with only its head and left lower extremity exposed. The foot, leg and thigh were clipped close before the electrodes were applied. Examinations were made on definite, predetermined days, from the eighth to the postoperative ninetieth day.

Electromyographic responses were of the following kinds: (1) no voltage response; (2) voltages from denervated voluntary muscle; (3) voltages from neurotized voluntary muscle and (4) voltages from normal voluntary muscle. (A slide was projected to show the wave form of these various voltages.)

Electromyography enables one, by recognition of fibrillation and nascent and normal action potentials, to differentiate accurately denervated, neurotized and normal voluntary muscle.

DISCUSSION

DR. R. P. MACKAY: In clinical neurology, fibrillary twitches are generally considered to indicate disease of the anterior horn cell. This report is interesting in that fibrillations occurred after section of the nerve; the anterior horn would seem to be eliminated as the source.

Were any of these animals curarized? If so, were the fibrillations abolished? Observation on this point would serve to indicate the site of origin of the stimulus to the twitch.

DR. L. J. POLLOCK: With regard to the clinical observation of fibrillation and its relation to the anterior horn, one might say it is unfortunate that the term fibrillation is used to describe what is seen when a nerve is severed as well as what is seen with lesions of the anterior horn. This fibrillation cannot be seen with ordinary light, but is seen with reflected light, usually under magnification. Fibrillation potentials are of the order of about 30 microvolts, whereas in the case

of the ordinary motor unit they were expressed in millivolts. What one sees in anterior horn disease is fasciculation. Fibrillation is also seen in anterior horn cell disease when the muscle becomes denervated. Curare has an effect on motor potential as well as on fasciculation. The rate of growth of the axon was studied in muscle by means of indication that a nerve fiber has arrived at the muscle, a high galvanic tetanus ratio being used as an indication. Where maturation occurs is unknown. Some may consider that restoration of function depends on maturation of the physical structure of the nerve, and others, that it depends on maturation of the myoneural junction; but it takes a long time after a nerve reaches muscle, sometimes one hundred and fifty days, before voluntary motor function returns. If one uses the motor unit potentials as an index, maturation is a far slower process than the growth of the axon—I think the latter is about 1.5 mm. per day. The discovery by electrodiagnosis that a nerve has grown into the muscle precedes by many days the discovery of conduction of impulses by electromyographic tracings.

Dr. James A. Fizzell: The only question which I wish to answer concerns the type of stimulation required to make the animals contract recently neurotized muscles. No electrical stimulation was used. Passive extension of the foot was tried at each examination after the thirty-third postoperative day, but it was discovered that application of pressure to the neuroma in the thigh was the easiest way to determine what voltages the muscles were capable of developing.

Book Reviews

The Master Hand. A Study of the Origin and Meaning of Right and Left Sidedness and Its Relation to Personality and Language. By Abram Blau, M.Sc., M.D., C.M. Research Monographs, no. 5, American Orthopsychiatric Association, Inc. Price, $4.50. Pp. 206. New York: American Orthopsychiatric Association, 1946.

This scholarly monograph records the work of ten years of a member of the faculty of New York University College of Medicine. It is one of the greater books that has appeared in the medical field within the lifetime of the reviewer. Putting it in another way, this book has a chance to live and to be pointed to by our successors as a landmark, which, when one thinks about it, is a distinction, whatever its merit, that may be awarded to few modern treatises of medicine. Whatever may be said of them, they are not built to endure.

The author, with his knowledge of art, history, philology and literature, to say nothing of medicine, has traced the development of ideas regarding preference for the use of the right or the left hand. His studies quite clearly demonstrate that theories of heredity and unalterable constitutional factors do not explain the problem, which is far too complex to be dispensed with as a simple instinct. He ties the study of handedness firmly to the considerably more fruitful consideration of laterality, or lateral dominance, by which the approach to a solution is broadened and, at the same time, is less charged, since no lateralization has become so emotionally colored and rigidly fixed in a neurologic pattern as has handedness. Thus, he is able to bring into the evidence such laterality of functions as leg kick, leg crossing, leg hop and hand clasping. A most important form of laterality which has heretofore received scant attention is the preferred direction of eye gaze, which, in this culture, is from left to right. Using an etching by Rembrandt (fig. 1), he illustrates beautifully the weighting of certain objects in the scene depending on the direction of eye gaze.

Dr. Blau, in his extensive researches on lateral dominance, has found with extreme rarity complete unilaterality of dextrality or sinistrality in all bilateral functions in a single person. Interestingly, the preference for left or right in the performance of some maneuver may vary in the same person with age and experience.

The preference for the right hand can be considered to have developed from the Bronze Age of prehistoric times. Previously there had been no signs of preferred group laterality. With the Bronze Age there was developed more complicated tools for one-sided use, and one-sided preference became relatively mandatory. These tools have been handed down to each succeeding generation, as has the experience with them.

Sinistrality has a relatively higher incidence in persons with certain character conditions, many of them unfavorable. As an example of a mild type, in the sport world a "lefty" is considered something of a "screwball." Sinistrality is more common in males than in females.

Left-handed parents unconsciously are models of sinistrality for their children, but the commonest type of sinistrality is probably the product of emotional contrariness in early childhood, accounting no doubt for its greater incidence among persons with antisocial disorders. The connotation that left handedness is bad or

good is not implied; here, again, is an instance in which it is to be emphasized that one is trying to understand behavior, and not to judge it.

In this monograph there is much interesting material on speech, reading reversals, mirror writing and the like. Stuttering, reading disability and developmental motor awkwardness have a type of psychoneurotic origin that is similar to that of negativistic sinistrality. As a conclusion to his extensive research, the author notes that a cerebral dominance is determined by function, and that people are left brained because they are right handed and, conversely, that they are right brained because they are left handed.

Children should be encouraged early to adopt right-sided habits, since this is a right-sided world. There is no danger in the retraining of children, as in the past had been thought to be the case, particularly in regard to stuttering. In children sinistrality may be a danger signal arising from emotional and environmental disturbances which should be corrected. In older persons it may be the insignificant relic of a neurosis in the past or part of a personality disturbance with a core of negativism. Sinistrality in many cases is only one symptom of an underlying disturbance of personality, and it should be superfluous to point out that the total disturbance should be dealt with, rather than a single symptom.

The demonstration that it is quite possible to train the nonpreferred hand to become the dominant one without emotional complications is of great importance; with this training there has been considerable experience after both world wars. What is required is careful and sympathetic instruction in the use of the other hand, with proper emphasis on the new directional orientations.

The book contains twelve chapters. It is printed on an excellent quality of dull, nonreflective paper. The twelve illustrations, in addition to the frontispiece, taken from the "Notebooks of Leonardo da Vinci" (truly an ambidextrous man) beautifully complement a masterful bit of publishing. Eight pages of bibliography and a careful index close a volume that should be in the library of all psychologists, neurologists, psychiatrists and medical historians and students, as well as in the possession of thoughtful teachers of children, to say nothing of him who has been concerned with his own left sidedness.

The Physical Background of Perception. By E. D. Adrian. Price, $3.25. Pp. 95 with 21 illustrations. London and New York: Oxford University Press, 1947.

This small volume makes exciting reading. The author, in his Wayneflete Lectures, has distilled the essence of his lifetime of work at Cambridge and with consummate skill has closed part of the gap between brain and mind. It being a small book, with only half a dozen chapters, one can read it with profit more than once in order to pick up some of the details of the author's thoughts.

The nervous impulse and the accompanying electrical change "are inseparable." Furthermore, "records of the electrical activity of the nerves leave no doubt that there is only one kind of change which can be conducted down a nerve-fibre, only one kind of impulse." From this most simple foundation a complex system is built up, by means of many different mechanisms. The number of nerve fibers and the frequency of their discharge are two aspects that are well understood. Intensity of stimulus is highly correlated with frequency of electrical discharge no matter what the number of units in simultaneous activity. Specialized sense organs are constructed to respond specifically to a variety of different stimuli without being injured by tremendous overloading. The function of the cerebral cortex is in a measure to act as a screen on which the incoming stimuli can be

projected. This is shown most clearly for visual stimuli, but holds also for auditory and possibly other systems. Pain, however, has no specific localization.

"The general property [of cerebral activity] is that which causes a sensory message or any change from the normal inflow of messages to set going the reactions which will extinguish the message or bring the balance back to normal. By reacting in this way the nervous system keeps the organism in harmony with its surroundings and its parts in harmony with one another." This concept may be carried from the simple process of withdrawing from a pinprick to the complex organization of human behavior for avoidance of hunger that "may lead to the planting of corn, to tractors and black markets and food conferences."

Electrical recordings indicate that consciousness is possible with only a fraction of the cerebral mechanism actively engaged. Furthermore, great areas may be removed from the cerebral hemispheres of rat or man without affecting consciousness, perception, memory or even the performance of skilled acts. "There may be concentration, diffusion, and interaction of the pathways on the way, but in simplest terms the mechanism for mapping events in the brain is no more than a very large number of more or less independent conducting units between the sensory surface and the cortex."

The cortex, however, requires the consideration of a new range of facts, such as those relating to recognition, memory and habit formation. "We can recognize a tune whether the pitch is high or low, and we can recognize a triangle or a letter whatever its size and whatever the position of the image on the retina. Now this must mean that there is no need for particular sensory endings or particular pathways to the cortex to be excited; for the recognition of the pattern all that seems to matter is that the excited regions should have certain general relations to one another. . . . The same principle seems to come in on the motor side as well as the sensory. We may learn a skilled movement by employing certain muscles and therefore certain groups of nerve cells in the motor area of the brain, but when we have learnt it we can carry out the movement with an entirely different set of muscles and nerve-cells—we can write our name with a pencil held between the toes when we have learnt to do it with our fingers. . . . The nervous system reacts to relations between stimuli and performs the task with any part of the motor system that is available."

Two main problems underlying the physical basis of perception are the power of abstraction and the nature of memory traces. These are considered in the final chapter. From the passages already quoted the reader may have his curiosity stimulated as to what Adrian has to say on these topics. The reviewer will not spoil the reader's fun by further quotation, particularly since he could not do justice to Adrian's thought without quoting the whole chapter.

The Nature of Deterioration in Schizophrenic Conditions. By David Shakow, Ph.D. Nervous and Mental Disease Monograph No. 70. Price, $3. Pp. 88. New York: Nervous and Mental Disease Monographs, 1946.

In this report, by the chief psychologist of the Worcester State Hospital, deterioration is defined as "functioning at a level below the subject's optimal performance due to an integral impairment of the organism rather than to such temporary or extrinsic factors as passing physical illness, fatigue, emotional disturbance or poor external conditions for performance." The author does not indicate how this definition was arrived at, although he does defend its value. What is meant by "an integral impairment of the organism" remains obscure, and why an emotional disturbance is considered temporary or extrinsic also is not clear.

The value of longitudinal studies is indicated, and the author deplores the limitation of the present report to cross sectional aspects.

The monograph is based mainly on the study of a group of 25 male schizophrenic patients, with additional data from larger groups. The undoubted importance of cooperation is discussed. Comparisons with "normal" control groups are made. The results obtained on the psychometric and experimental measures are evaluated in the light of four types of variables: (1) general intelligence, (2) apperception, (3) conation and (4) motor ability and learning, with the addition of two other types of variables: (1) gross behavior variables and (2) certain descriptive variables, such as age and education.

The section on results contains an interesting résumé of previous literature on the subject. The results in the present study seem to indicate that the schizophrenic group is most affected unfavorably in tests involving conceptual thinking. The term "conceptual thinking" was taken from a system of classification developed by Dr. Shakow for the items in the Stanford-Binet scale. This system is described by the author in a footnote, and it seems to this reviewer that it should have been given more prominence. Much attention is given to the importance of a higher vocabulary score, on which the author casts doubt. Findings in the Rorschach test are in general accord with the results of previous investigators. Significant differences were found in the Army Alpha and Kent-Rosanoff tests and in the series of motor and learning tests. In the accessibility and interruption experiments, the larger group study was used, with the finding that the schizophrenic subject's behavior is more superficial and less goal determined.

A high correlation was found between clinical deterioration ratings and the variables studied.

In his discussion, the author describes the picture of schizophrenia in an individual patient, based not only on the data from his study but on the observations of other writers as well. On the basis of a theory of levels of deterioration, a chart of relations of such levels is presented. This is interesting but suffers from the disadvantages of using a hypothetic division into stages and special descriptive terms.

The monograph is well summarized in the final chapter.

This study is instructive but does not contribute much that is new to the understanding of schizophrenia. The nature of deterioration remains obscure, and some other approach seems necessary to clarify this difficult subject.

Manual of Psychological Medicine. By A. F. Tredgold, M.D., F.R.C.P. Price, $5. Pp. 298. Baltimore: The William Wood & Company, 1943.

This book is, in effect, a condensed textbook of psychiatry. It was written in Britain at the height of the war, at a time when the problems of psychiatry for civilian and soldier alike were sharply focused. The author explicitly states that its purpose was to provide for the needs of the civilian practitioners, members of the army recruiting boards and medical officers, who were currently confronted with the bulk of problems in the domain of "psychologic medicine." It is within these limitations of scope and purpose that this work must be viewed. It should not be conceived of as adequate for the psychiatrist or the serious student of psychiatry.

The text follows generally the pattern of most comprehensive works in psychiatry. For purposes of description, it may be divided into three parts, of which the middle section constitutes the bulk. The first portion includes chapters on the normal mind, nomenclature and classification of abnormality, causation and general symptomatology. The larger (middle) section comprises approximately

eighteen chapters devoted to a condensed, but relatively inclusive, survey of the various mental illnesses. The final chapters bring together such diverse topics as special forms of treatment, legal relationships, sociologic considerations and procedures and duties of the medical practitioner. The description of legal relationships may be of particular interest to the reader who seeks familiarity with British law as it relates to psychiatric problems.

The author's system of classification is of some interest. Three primary categories are established, within which all individual mental illnesses are further classified. The first of these main groupings is "mental disorder," which encompasses conditions of which the most significant feature is a functional disturbance of mind. The psychoneuroses and certain of the psychoses fall into this group. Another basic category is "mental decay," in which are to be found conditions characterized chiefly by permanent degeneration of mind, examples of which are presenile dementia and senile dementia. All mental states not included in the areas of "disorder" and "decay" in which the common factor of arrested or incomplete development of mind is present are categorized by the author as "mental defect."

Particularly noteworthy throughout are the clarity and adequacy with which the author has handled the description of mental disturbances in their development and symptomatology. This has been done despite serious limitations of space. Also effective and valuable are the sections on mental defect, a subject which the author has in the past dealt with more exhaustively. Despite the praiseworthy aspects of this work, there are serious failings which detract considerably from its merit. Chief among these is the unfortunate, but consistent, fashion in which the author emphasizes the concepts of psychopathic predisposition, psychopathic inheritance, poor mental stamina, and the like, as fundamental etiologic factors in almost all psychopathologic states. He evidently is unaware of, or unwilling to include, modern dynamic concepts in his orientation. The effect is to create an impression that no other interpretation or understanding is possible. This lends to the author's concept of psychiatry a fatalistic and therapeutically nihilistic attitude, which can do little to correct an already tragic prevalence of defeatism among sections of nonpsychiatric medical opinion.

The book is not recommended for psychiatrists or medical students.

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